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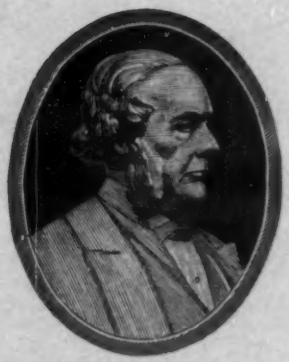

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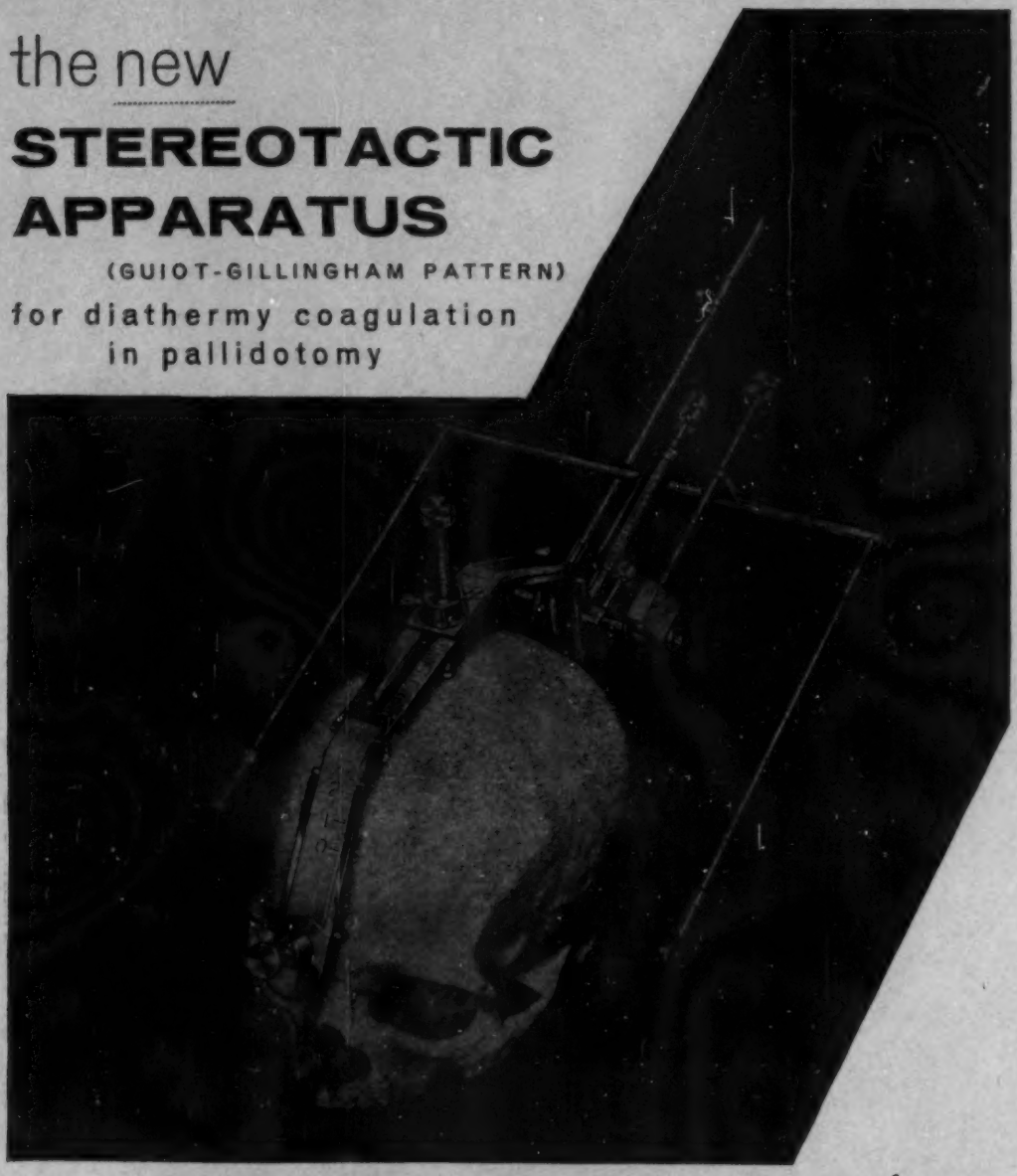

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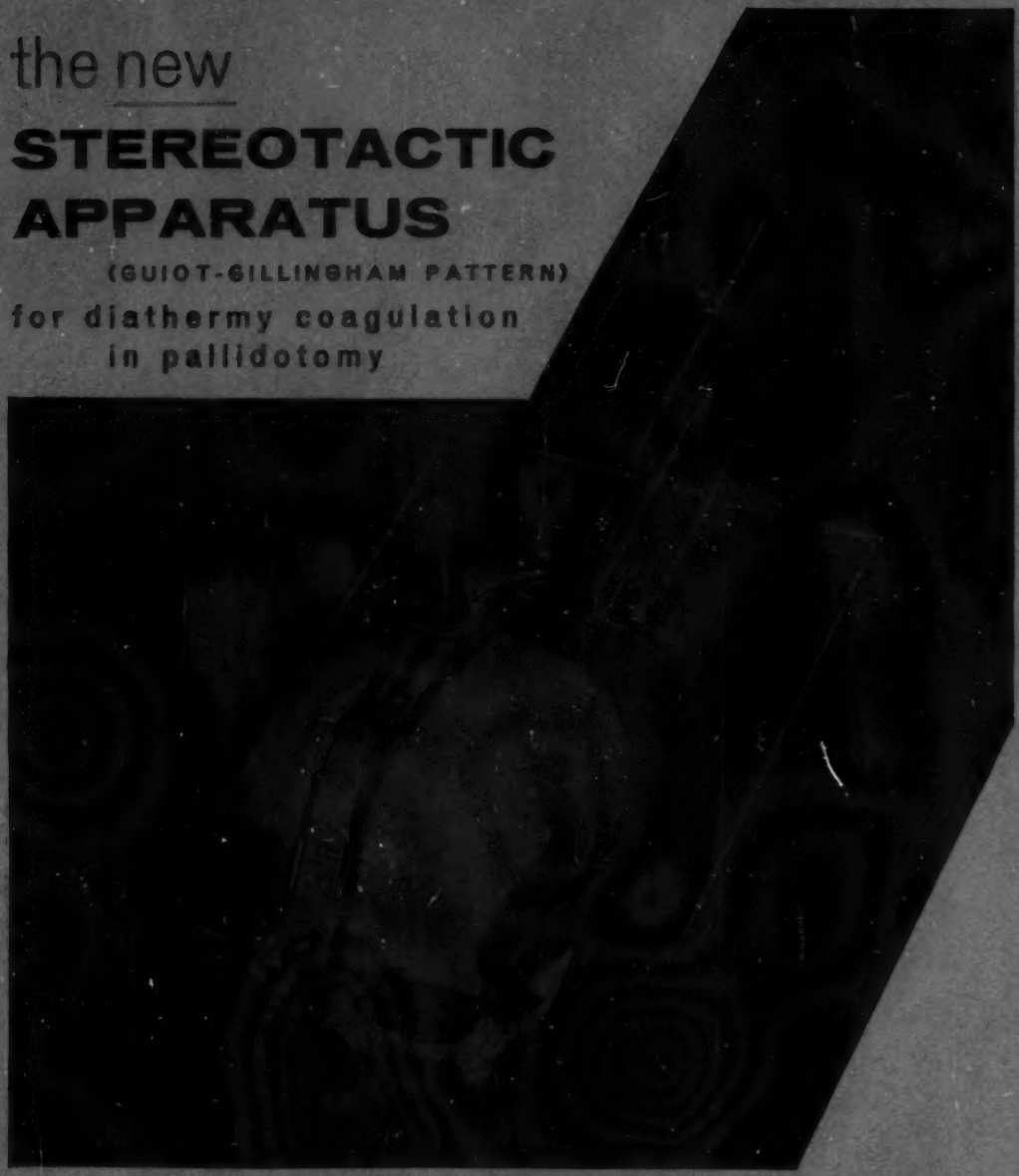

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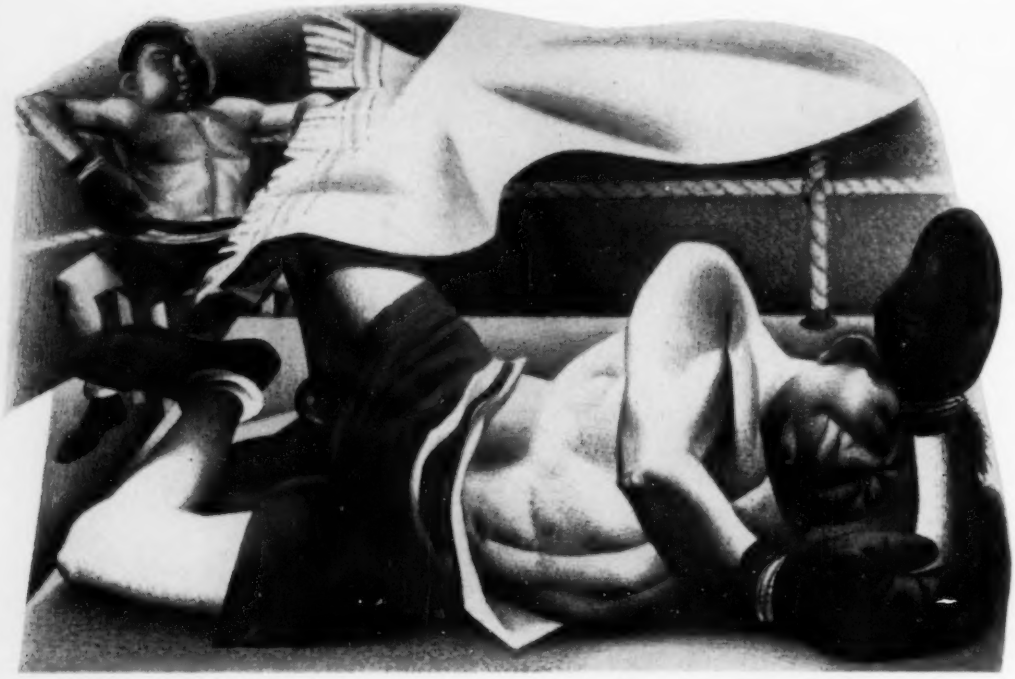

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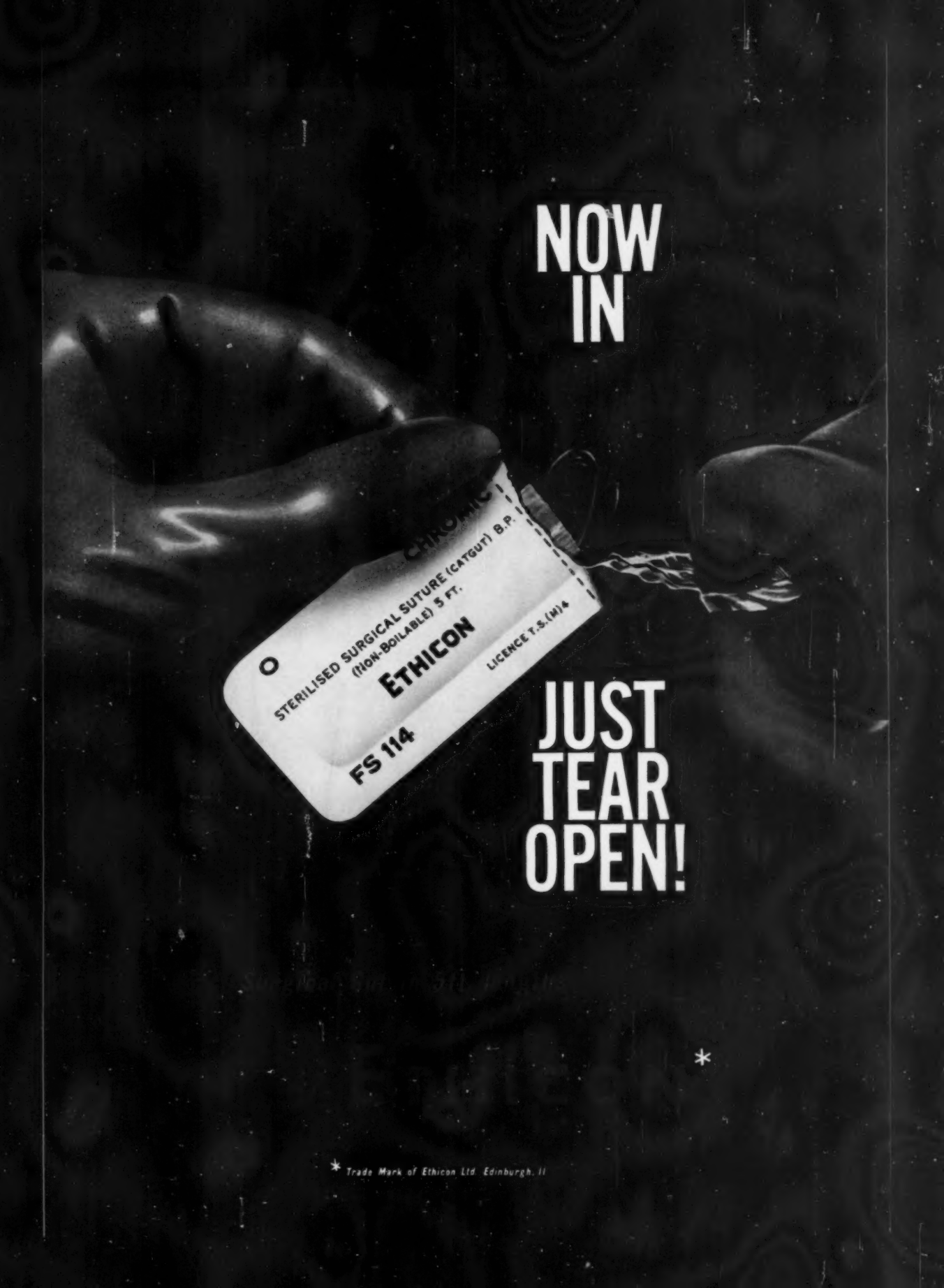
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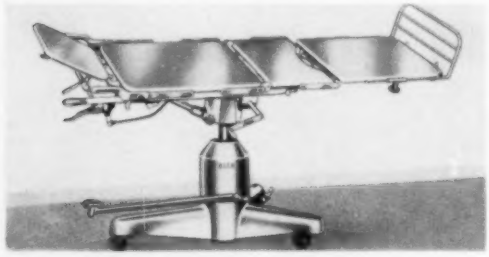

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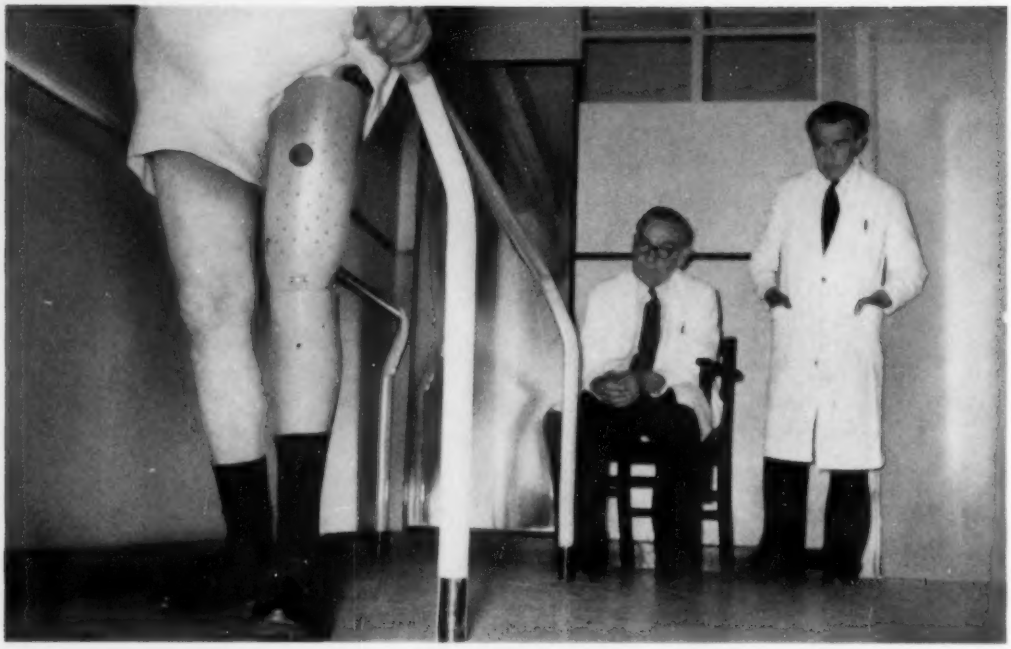

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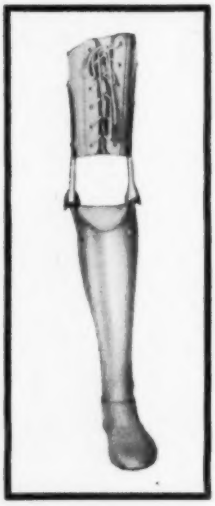

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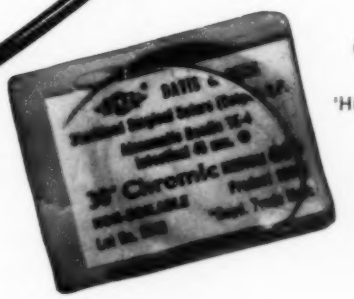

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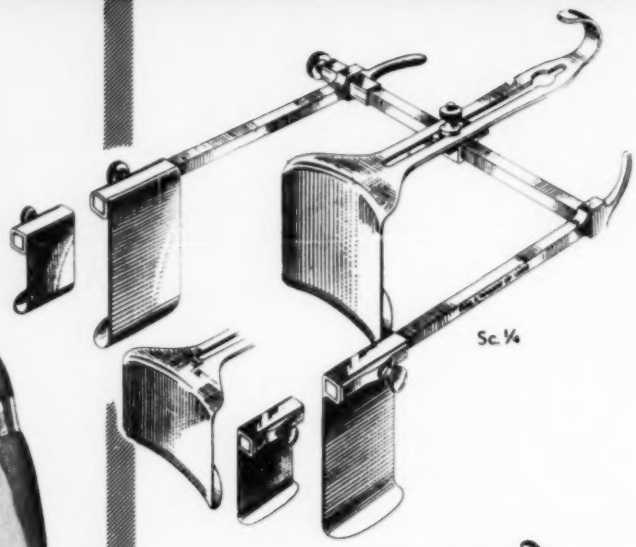

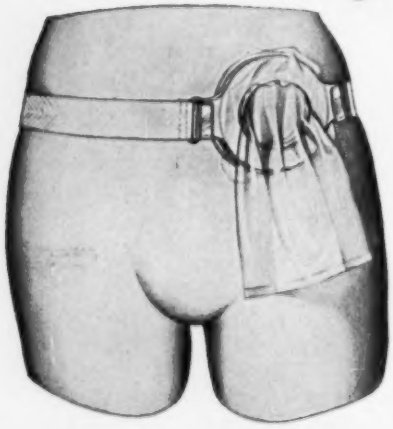

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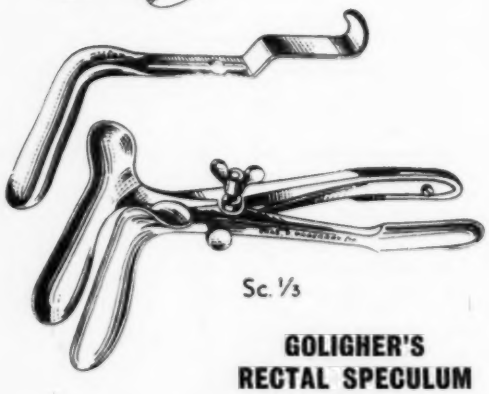

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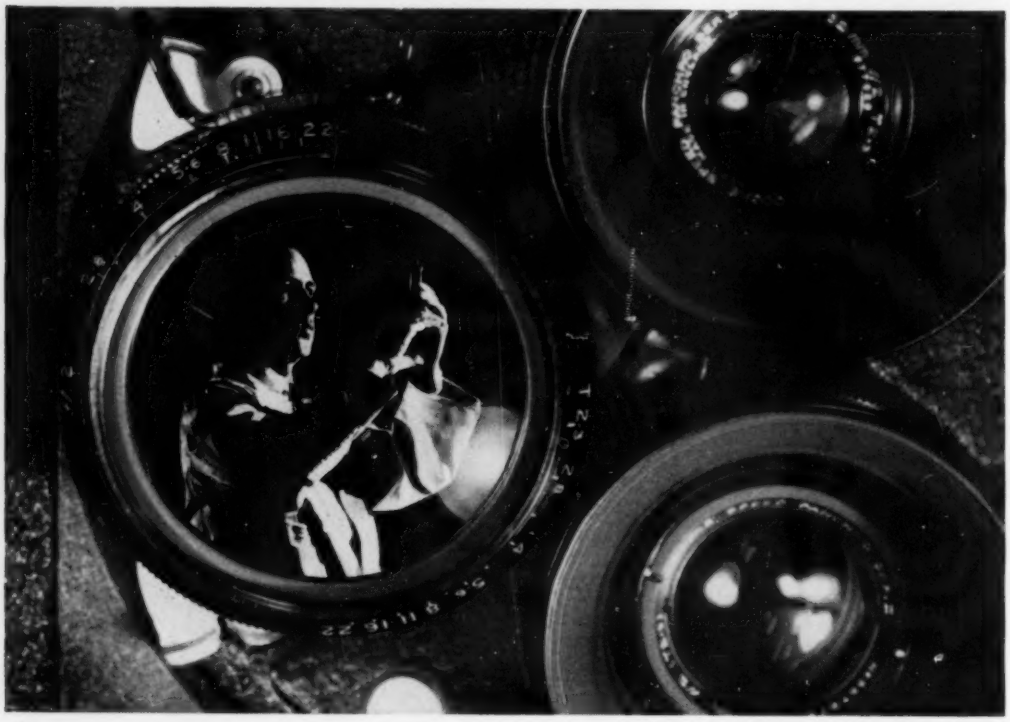

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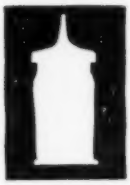

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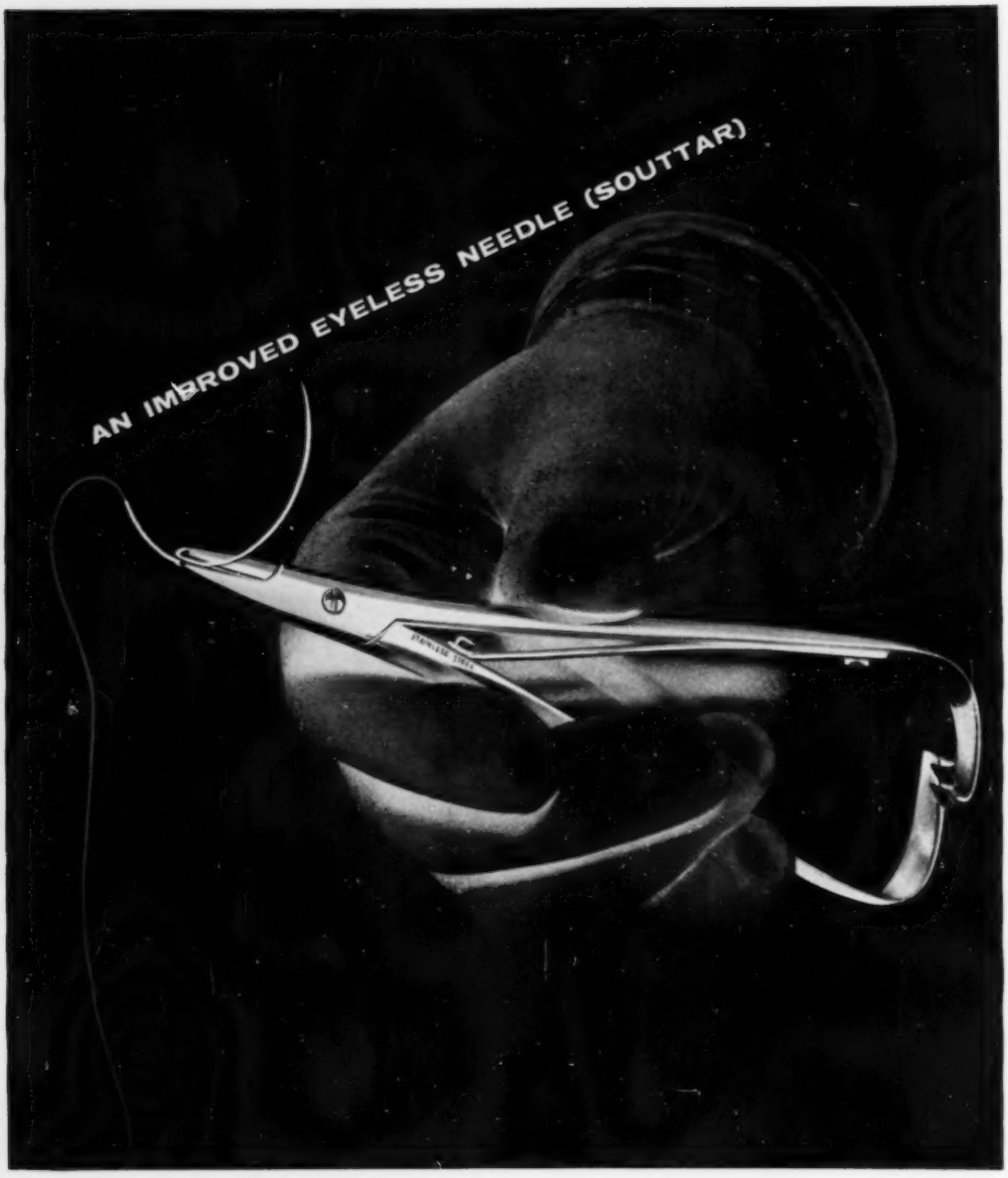

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THE
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VOL. XLIX SEPTEMBER, 1961 NO. 214

THE SURGICAL PATHOLOGY OF GASTRO-ŒSOPHAGEAL CARCINOMA

BY O. G. DODGE

DEPARTMENT OF PATHOLOGY, UNIVERSITY OF SHEFFIELD

RECENT advances in surgical techniques have made the gastro-œsophageal region much more readily accessible, and have drawn attention to the lesions affecting this hitherto neglected area. Surveys of gastro-œsophageal carcinoma from the surgical viewpoint have been made by Allison (1946), Allison and Borrie (1949), Sweet (1952), Nuboer (1954), and Carey and Clagett (1955). Usually, however, carcinoma of the gastro-œsophageal region has been considered only incidentally in surveys of gastric or

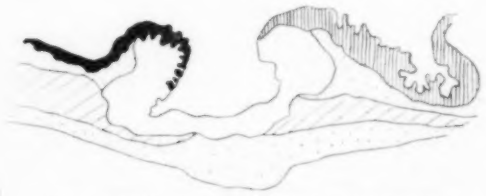

FIG. 116.—Diagram of a longitudinal section through an ulcerated gastro-œsophageal carcinoma. Key: Œsophageal epithelium, black; gastric mucosa, vertically shaded; muscle, diagonally shaded; tumour, unshaded; other tissues, stippled. The same markings are used in *Figs.* 117, 118, 120, and 121.

œsophageal carcinoma. The only detailed survey of the pathology of gastro-œsophageal carcinomas as such is the account of 65 resected tumours by McPeak and Warren (1948). The present study, based on 87 cases of surgically excised gastro-œsophageal carcinomas, is complementary to their work.

MATERIALS AND METHODS

All the tumours studied were surgically resected. Post-mortem digestion makes necropsy material from this region almost useless for histological study. Longitudinal whole-tumour sections, multiple in many cases, were available for study. Routine histological stains, including periodic acid-Schiff and reticulin techniques, were used.

CLASSIFICATION OF TUMOURS

Table I shows the anatomical and histological groups into which the carcinomas fell, and the number in each. Carcinomas which lay more or less equally to either side of the gastro-œsophageal junction are classed as 'junctional'. The following points are worthy of note:—

1. Only 7 of the 25 carcinomas situated mainly in the œsophagus were squamous carcinomas; 11 were adenocarcinomas and 5 were carcinomas of mixed histological type.

2. No squamous carcinomas recurred in the junctional or mainly intragastric groups; 55 of these 62 tumours were adenocarcinomas.

3. Anaplastic carcinomas occurred on both sides of the junction.

4. Carcinomas of mixed histological type are not uncommon in the gastro-œsophageal region.

ADENOCARCINOMA

The *mainly intragastric* adenocarcinomas showed the usual features of carcinoma of the cardia. *Fig.* 116 is a diagram of a longitudinal section through

FIG. 117.—A carcinoma straddling the gastro-œsophageal junction. Relatively superficial growth in œsophagus, but deeply infiltrative in stomach. Markings as in *Fig.* 116.

one of these tumours, showing the undermining of both gastric and œsophageal epithelium by carcinoma. The *junctional* adenocarcinomas, lying astride the gastro-œsophageal junction, usually presented as a fungating or ulcerated fleshy tumour in the stomach, and as an annular infiltrative constricting mass in the lower œsophagus. The contrast between the morphology of the tumours in the two organs is brought out in *Fig.* 117. *Fig.* 118 shows a deeply infiltrative tumour astride the junction with a small outlying tumour

Table I.—SITUATION AND HISTOLOGICAL STRUCTURE OF 87 GASTRO-ŒSOPHAGEAL CARCINOMAS

HISTOLOGICAL TYPE	ANATOMICAL SITUATION			TOTAL
	Mainly Intra-œsophageal	Junctional	Mainly Intragastric	
Adenocarcinoma	11	16	39	66
Squamous carcinoma	7	0	0	7
Anaplastic carcinoma	2	0	5	7
Mixed carcinoma	5	1	1	7
Total	25	17	45	87

nodule beyond the proximal margin of the main mass (*Fig.* 119). These satellite nodules were commonly seen in the œsophagus, and illustrate the difficulty of ensuring adequate excision of these tumours. In this case there was a local recurrence 18 months after resection.

The *mainly intra-œsophageal adenocarcinomas*, 11 in number, comprised 4 cases in which a carcinoma clearly of gastric origin had spread proximally into the lower or even to the mid-œsophagus, and 7 cases in which the adenocarcinoma lay entirely within the anatomical confines of the œsophagus. In at least 6 of

FIG. 118.—A carcinoma astride the gastro-œsophageal junction, with a 'satellite' tumour nodule beyond the proximal margin of the main tumour mass. Markings as in *Fig.* 116.

these 7 cases the adenocarcinoma had arisen in a 'lower œsophagus lined by columnar epithelium' (Barrett, 1957). One of the 4 gastric adenocarcinomas with extensive intra-œsophageal spread is shown in *Fig.* 120; at the proximal margin of resection, tumour was present in the submucosa beneath apparently normal surface epithelium. This emphasizes the point that microscopy often shows spread into the œsophagus well beyond the tumour margin as apparent at operation. This, and the presence of satellite nodules, probably account for the

FIG. 120.—A carcinoma extending across the gastro-œsophageal epithelium and extending far into the œsophagus, up to the margin of resection, beneath largely intact epithelium.

frequency of local recurrence following œsophago-gastrectomy.

SQUAMOUS CARCINOMA

Only 7 squamous carcinomas were found involving the gastro-œsophageal junction, compared with 66 adenocarcinomas. Not only were they much less frequent, but the extent of their spread into the stomach was much less than that of adenocarcinoma into the œsophagus. These were all annular constricting tumours, centred in the lower œsophagus. In each case only a narrow rim of tissue at the cardiac end of the stomach was involved. *Fig.* 121 is a diagram of the distal end of one of these tumours, showing its abrupt rounded edge. It appears indeed as if there is a barrier to further extension of œsophageal carcinomas into the stomach.

One case of squamous carcinoma showed several points of interest. This patient originally had a carcinoma of the middle third of the œsophagus. The tumour was excised and a gastro-œsophageal anastomosis performed. Microscopical examination revealed a well-differentiated squamous-cell carcinoma. Above the proximal edge of the tumour, however, an area of abnormal epithelium was present, and microscopy showed that this was an area of intra-epithelial carcinoma in the œsophagus (*Fig.* 122). This area was separated from the main tumour by normal squamous epithelium. Six years later,

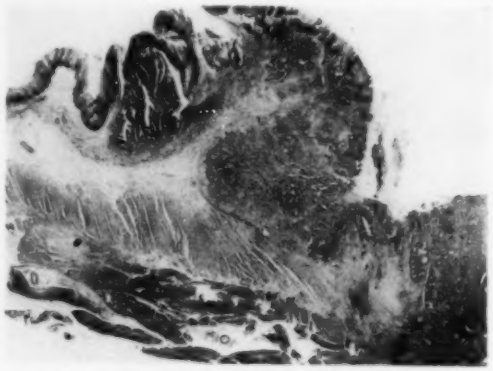

FIG. 119.—Proximal edge of tumour shown in *Fig.* 118. Satellite nodule in œsophageal submucosa. H. and E. (× 3·25.)

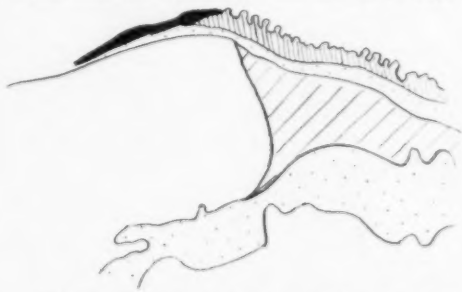

FIG. 121.—Distal margin of an œsophageal squamous-cell carcinoma. Abrupt margin of tumour at gastro-œsophageal junction. Markings as *Fig.* 116.

dysphagia returned. At operation recurrent tumour was found at the anastomosis. This was excised, and a new gastro-œsophageal anastomosis performed. *Fig.* 123 shows part of the excised anastomosis. At this artificial gastro-œsophageal junction, there was considerable mingling of squamous and glandular epithelium, instead of the usual sharp division, and there was a more marked infiltration of the gastric mucosa by invasive squamous carcinoma than was observed in any of the other squamous carcinomas in this series.

The relative resistance of the stomach to invasion by carcinoma of the adjacent œsophagus recalls the rather similar resistance of the duodenum to invasion by carcinoma of the stomach. In neither case is resistance absolute; microscopical examination may reveal invasion beyond the apparent tumour margin. The comparative freedom with which the tumour in this last case infiltrated across an *artificial* gastro-œsophageal junction suggests that some natural barrier may have been removed when the original junction was excised.

ANAPLASTIC CARCINOMA

Five of these 7 carcinomas lay mainly in the stomach, and 2 mainly in the œsophagus. Four of the 5 mainly intragastric growths were centred on the proximal third of the stomach, involving the œsophagus at their upper margins. The fifth was a scirrhous carcinoma of the entire stomach, invading the œsophagus. These 5 tumours were clearly of gastric origin. In 1 case intramural invasion of the œsophagus extended up to the level of the root of the lung. One of the mainly intra-œsophageal carcinomas resembled an undifferentiated transitional-cell carcinoma (*Fig.* 124), with ribbons and 'garlands' of tumour cells separating pools of serous secretion. This pattern is like that seen in some nasal carcinomas (Ringertz, 1938), and this tumour may have arisen in an œsophageal mucous gland. This patient was 26 years old, the youngest in the series.

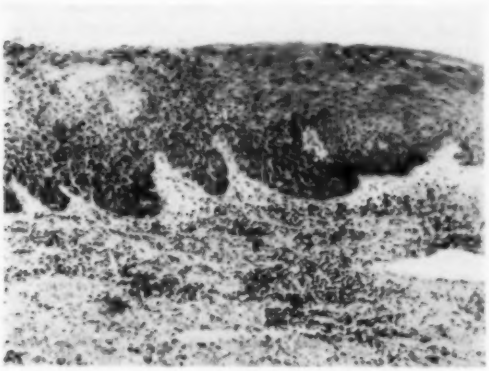

FIG. 122.—Intra-epithelial carcinoma in œsophagus. H. and E. (× 32·5.)

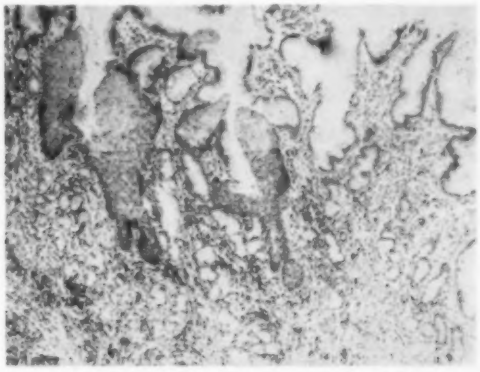

FIG. 123.—Mingling of gastric and œsophageal epithelium at a gastro-œsophageal anastomosis. H. and E. (× 24.)

MIXED CARCINOMA

Seven carcinomas showed various types of mixed histological pattern. Two of these were adeno-carcinomas with areas of squamous metaplasia. Two had the structure of muco-epidermoid carcinoma, and had probably arisen in œsophageal mucous glands or their ducts. A further 2 carcinomas showed some topographical separation between their two components, and we may have had a double histogenesis from both œsophageal and gastric epithelium. The seventh case was an undoubted 'collision carcinoma'—here, a gastric adenocarcinoma and an œsophageal anaplastic carcinoma had grown together to form a single tumour mass. These cases are being fully described elsewhere (Dodge, 1961).

From the clinical viewpoint, these mixed tumours may give cause for confusion since successive biopsies may reveal adenocarcinoma and squamous-cell carcinoma, for example. To the pathologist these mixed carcinomas illustrate the many potentialities for differentiation possessed by the generative cells of the alimentary tract, and confirm what has already been described at other sites, the great variety of tumour types encountered at interepithelial junctions.

THE FREQUENCY OF LOCAL RECURRENCE AND METASTASIS

The considerable technical difficulty of ensuring complete removal of gastro-œsophageal carcinomas means that local recurrences are not uncommon after excision. In this series there was evidence (clinical or necropsy) of local recurrence in 14 out of 66 adenocarcinomas, and in 2 out of 6 carcinomas of mixed type. None of the squamous-cell or anaplastic carcinomas showed local recurrence.

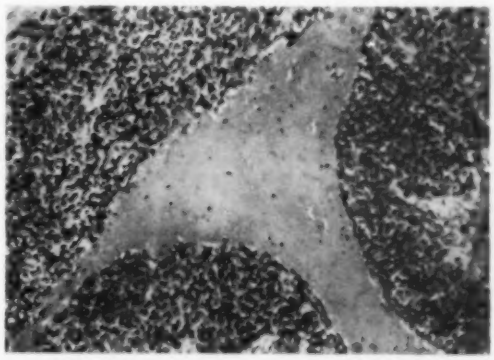

FIG. 124.—Anaplastic carcinoma of transitional-cell type, with central pool of necrosis. H. and E. (× 60.)

Metastases are known to have occurred (on clinical or necropsy evidence) in 15 cases (10 adeno-carcinomas, 1 squamous carcinoma, 1 anaplastic carcinoma, and 3 mixed carcinomas). Twenty of the 87 cases have come to necropsy. In 15 of these there was no evidence of metastatic tumour.

The metastases were surprisingly limited in distribution. The commonest sites were intra-abdominal lymph-nodes (4 cases), mediastinal and cervical lymph-nodes (3 cases each), ribs and chest wall (3 cases), and liver (3 cases). The adrenals were involved in 1 case and the lungs in 2 cases. No other remote metastases were encountered, and many of the secondaries found may have been the result of local invasion rather than of true metastasis.

Survival rates are nevertheless low: 21 out of the 87 cases died within one month of operation; 16 are known to have survived one year (4 squamous, 1 anaplastic and 11 adenocarcinomas); only 1 case (a squamous carcinoma) is so far known to have survived five years. Many of the later patients in the series, however, are still alive, and may well survive five years from the time of operation. As they represent the latest patients in the group, they should benefit from the increased experience of surgeons in the selection of cases and in operative technique.

DISCUSSION

The Incidence of Gastro-œsophageal Carcinoma.—It is difficult to estimate accurately the frequency of tumours involving the gastro-œsophageal junction. These tumours are usually classed as carcinomas of the œsophagus or of the stomach. The present writer's calculations (*see* APPENDIX) indicate that the frequency of these two groups must together be at least as great as that of œsophageal cancer as a whole.

The Frequency of Œsophageal Invasion by Gastric Carcinoma.—It is well known that the œsophagus, unlike the duodenum, offers no resistance to the spread of gastric carcinoma. Zinninger and Collins (1949) found invasion of the œsophagus in 5 out of 6 cases of carcinoma of the cardia. The present series illustrates the extensive intra-œsophageal spread of carcinomas of gastric origin. Analysis of 53 resected adenocarcinomas of the stomach involving the cardiac region showed that 31 (58·5 per cent) of them had invaded the œsophagus.

The Frequency of Gastric Invasion by Œsophageal Carcinoma.—Cases of œsophageal carcinoma invading the stomach are mentioned by Allison and Borrie (1949) and reviewed by Willis (1952). The present series, however, shows that, of carcinomas involving the gastro-œsophageal junction, the great majority clearly originate from the gastric side (66 adenocarcinomas—39 of them mainly intragastric—as against 7 squamous-cell carcinomas of the œsophagus). If œsophageal carcinoma were as liable to transgress the gastro-œsophageal junction as is gastric carcinoma, a higher proportion of squamous-cell carcinomas involving the gastro-œsophageal junction would be expected than was observed in the present series.

The extent of gastric invasion by squamous-cell carcinoma was also very limited. In no case did it approach the extensive infiltration of the œsophagus seen in many of the adenocarcinomas, and in most cases the distal edge of the tumour was smooth and sharply defined, as shown in *Fig.* 121.

McPeak and Warren (1948) gave a detailed account of the histology of 65 cases of carcinoma of the cardio-œsophageal junction (57 of them surgical resections). They found only 2 cases of epidermoid carcinoma, but 55 cases of adenocarcinoma of various types. At least 32 of their tumours were considered to be gastric adenocarcinomas invading the œsophagus. The present series shows a similar heavy preponderance of gastric over œsophageal carcinomas.

These observations indicate that the gastro-œsophageal junction offers at least a partial resistance to the spread of œsophageal carcinoma into the stomach (recalling the more striking resistance of the duodenum to invasion by gastric carcinoma). There is, on the contrary, no obvious barrier to the invasion of the œsophagus by gastric carcinoma.

Aetiological Factors.—In the present series of 87 cases there was no recorded family history of cancer, no known cases of syphilis or cirrhosis, and no recorded histories of high alcohol intake, or of previous trauma to the œsophagus or stomach. Five cases, however, had a history indicating previous œsophageal or gastric disease. Three patients had a history of hiatus hernia, 1 case had a chronic peptic ulcer, and 1 a submucosal cyst at the cardia.

Histogenesis.—The mucous neck cells of the gastric glands are the stem cells of the gastric mucosa and the cells of origin of gastric carcinoma (Ivy, 1955). Intestinal metaplasia of the gastric mucosa may well be a precancerous lesion (Morson, 1955b). It was seen in sections of only 3 of the 66 adenocarcinomas—this may reflect the fact that the frequency of intestinal metaplasia falls from the distal to the proximal end of the stomach (Morson, 1955a). Œsophageal squamous carcinoma arises from the basal cells of the squamous epithelium. Steiner (1956) has shown that even very early œsophageal carcinomas are invasive. A long in situ phase is not usual in these growths, but intra-epithelial carcinoma of the œsophagus was seen in one case of the present series (*Fig.* 122) and has been described occasionally (O'Gara and Horn, 1955).

The histogenesis of the intra-œsophageal adenocarcinomas is controversial, but evidence is presented elsewhere (Dodge, 1960) that many of these tumours arise in a lower œsophagus lined by columnar epithelium, as described by Barrett (1957).

The histogenesis of the mixed carcinomas raises the question of the unicellular and field concepts of carcinogenesis; some of these tumours may arise, at least in part, from the mucous glands of the œsophagus, or from their ducts. A similar range of mixed histological patterns is encountered at other inter-epithelial junctions, such as the cervix uteri (Hamperl and Hellweg, 1957) and the anorectal region (Kay, 1954).

CONCLUSIONS

The majority of carcinomas involving the gastro-œsophageal junction are adenocarcinomas of gastric origin (66 cases out of 87 in the present series).

The gastro-œsophageal junction offers no resistance to the spread of these tumours into the œsophagus, which is often widely invaded. Outlying nodules of gastric carcinoma and intramural spread within the œsophagus may be present to an extent not apparent macroscopically. In these respects the gastro-œsophageal and gastroduodenal regions afford a remarkable contrast.

Squamous-cell carcinomas of the œsophagus involving the gastro-œsophagus region are very much less frequent than gastric adenocarcinomas (7 cases against 66 in the present series). They show, on the whole, a very limited degree of extension into the stomach, which appears to offer an unfavourable environment for invading œsophageal carcinoma. The apparent resistance of the stomach to the spread of œsophageal carcinoma recalls that of the duodenum to the spread of gastric carcinoma.

Anaplastic carcinomas involving the gastro-œsophageal region were as frequent, in this series, as squamous-cell carcinomas (7 cases of each). Two adenocarcinomas with areas of squamous metaplasia, 2 muco-epidermoid carcinomas, 2 carcinomas each with two distinct components, and 1 collision carcinoma were encountered.

APPENDIX

The Incidence of Gastro-œsophageal Carcinoma.—Carcinoma of this region is included in statistical surveys either with carcinoma of the œsophagus or of the stomach. To gain some idea of the incidence of tumours of the junctional area, it is necessary to add together the numbers of carcinomas involving the lower third of the œsophagus and of gastric carcinomas involving the cardiac region of the stomach. The proportion of œsophageal carcinomas involving the lower third is very variously assessed. Figures range from 18·3 per cent (Shedd, Crowley, and Lindskog, 1955) to 53·8 per cent (Watson, 1933). In the most recent large series, (Terracol and Sweet, 1958) 42·5 per cent of œsophageal carcinomas involved the lower third—adenocarcinomas were excluded from this estimate, which is probably the most accurate now available.

Table II.—The Relative Incidence of Carcinomas at Various Anatomical Sites (Figures from Harnett, 1952) Compared with that of Carcinoma of the Lower Œsophagus plus Cardia of Stomach (Author's Estimate)

Site	No. of Cases	Ratio to Incidence of Œsophageal Cancer (=100)
Œsophagus	470	100
Stomach	1405	300
Colon	892	189
Bladder	423	90
Tongue	287	61
Lower œsophagus plus cardia of stomach	—	111

The gastric carcinomas that may involve the œsophagus comprise those situated in the region of the cardia and those involving the whole stomach. A series of 345 cases of gastric carcinomas treated surgically by some form of gastrectomy at the Sheffield Royal Infirmary in the 10-year period 1949–58 was analysed according to the site of lesion. The tumours confined to the cardia, plus the tumours which involved the whole stomach (or the major part, including the cardia), accounted for 80 out of the total 345—23·2 per cent.

Harnett (1952) has shown that in Great Britain gastric and œsophageal carcinoma occur in the proportion of 3: 1. If we arbitrarily take the incidence of gastric and œsophageal carcinomas as 300 and 100, and if we estimate that 23 per cent of gastric carcinomas (i.e., 69 out of 300) and 42·5 per cent of œsophageal carcinomas may involve the gastro-œsophageal junction, then this gives a total of 69 + 42·5 = 111·5 for the total incidence of carcinoma of the lower œsophagus and cardia as a whole. If this is a fairly reliable estimate, then carcinoma of this region is commoner than carcinoma of the œsophagus as a whole, and also greater than that of carcinoma of the tongue or bladder (*Table II*).

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FACTORS AFFECTING RESPIRATORY CAPACITY IN PATIENTS UNDERGOING ABDOMINAL SURGERY

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THE problem of pulmonary collapse and consolidation following abdominal operations is a persistent one and seems to have improved to only a small extent in recent times. The advent of relaxant anæsthesia may even have made the problem worse, in so much as the cough reflex is often suppressed or minimized during the first critical hours of the post-operative period, when the seeds of collapse and consolidation are sown.

In order to assess the extent of the problem at the present time, measurements of respiratory capacity have been made throughout the operative period on a series of patients undergoing a variety of operations.

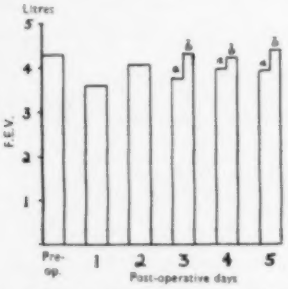

FIG. 125.—Histogram showing no considerable changes in breathing capacity after an operation for inguinal hernia. Breathing capacity is also compared in two different positions.

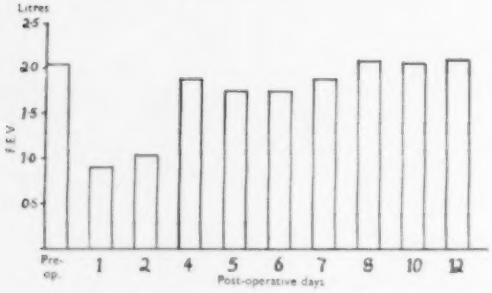

FIG. 127.—Histogram showing the considerable reduction in breathing capacity after pylorectomy despite the insignificance of the operative procedure. The delayed reduction at 5 and 6 days is also shown.

An attempt has also been made to define some of the more important factors leading to collapse of the lung and emphasis has been placed on patients undergoing abdominal operations.

Respiratory function can be assessed in three ways: (1) By *time-volume* studies, recording the maximum ventilation possible in a certain time period; (2) By *pressure-volume* measurements recording lung compliance (Attinger, Goldstein, and Segal, 1956); (3) By analysing the *gaseous exchanges* in the lung from blood and ventilatory samples. The first group of tests is the simplest and the most widely used (*Brit. med. J.*, 1958), and was considered to be adequate for the needs of the present investigation.

TECHNIQUE

The forced expiratory volume, which is the volume of gas expired as rapidly and as forcibly as possible over a stated time period (Gandevia and Hugh-Jones, 1957), was used as an index of respiratory capacity in these studies and was measured over a period of 1 sec. ($F.E.V._{(1)}$). Kennedy (1953) and Tiffeneau, Bousser, and Drutel (1949) have shown that there is a close correlation between the $F.E.V._{(1)}$ and the maximum breathing capacity; Gandevia, Hume, and Prime (1957) concluded that, from the practical clinical point of view, measurement of the F.E.V. is a sensitive indication of respiratory capacity changes

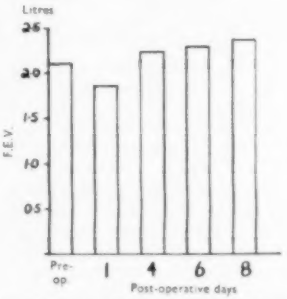

FIG. 126.—Histogram showing the mild and transient reduction in breathing capacity after mastectomy.

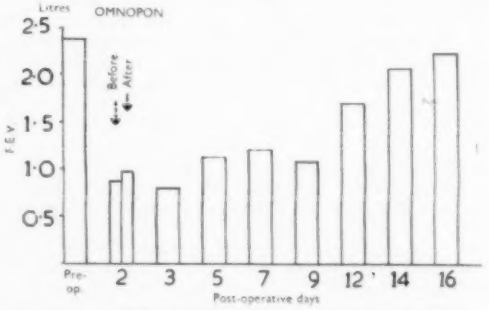

FIG. 128.—Histogram showing severe reduction in breathing capacity after partial gastrectomy. A delayed reduction at 9 days is shown. The improvement in breathing capacity after an injection of omnopon in the immediate post-operative period is also shown.

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and nothing of value is gained by doing measurements of intrathoracic pressure.

The apparatus used for the measurements was a sensitive spirometer recording on the drum of a fast-moving kymograph of the type designed by Bernstein, d'Silva, and Mendel (1952).

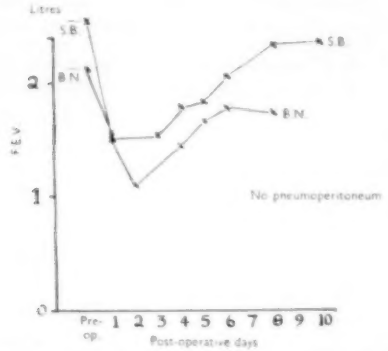

FIG. 129.—Graph showing the reduced breathing capacity of two cases undergoing appendicectomy. Return to normal occurs after 7 and 5 days respectively.

The tests were all performed with the patients sitting in bed, as change in position leads to alteration in respiratory effect (*see below*). The patient used a rubber mouthpiece attached to the breathing tube and learned to do the test before operation. Following this, no learning effect was observed, confirming the work of Friend (1955). At each sitting at least three forced expiratory volumes were recorded and the maximum of the three used as the result. Each patient was tested before operation and daily in the post-operative period until the patient was discharged from hospital, or the F.E.V. returned to normal.

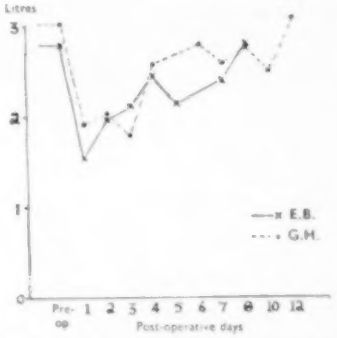

FIG. 130.—Graph showing the reduction in breathing capacity in two cases of partial gastrectomy. Return to normal is on the eighth and twelfth day respectively.

MATERIAL

A total of 59 cases were included in this survey. Consecutive cases on one unit were studied over a certain time period and there was no selection.

Abdominal operations show a much higher incidence of post-operative pulmonary collapse than

Table I.—CASES STUDIED BY SPIROMETRY, SHOWING MAXIMUM POST-OPERATIVE REDUCTION IN F.E.V.$_{(1)}$ AND DAY OF RETURN TO NORMAL

GROUP	OPERATION	NUMBER	MAXIMUM POST-OPERATIVE REDUCTION (PER CENT OF PRE-OPERATIVE)	NUMBER RETURNING TO NORMAL F.E.V. BY NINTH DAY
Upper abdominal	Gastrectomy	21	51·7	5
	Pylorectomy	3	52·0	1
	For post-gastrectomy syndrome	2	57·0	—
	Gastropexy	1	63·0	1
	Local gastrectomy	1	76·0	1
	Cholecystectomy	6	64·0	3
	Gastro-enterostomy	2	56·0	—
	Laparotomy	2	46·5	1
		38 (total)	54·6 (average)	10 (26·3 per cent)
Lower abdominal	Paramedian appendicectomy	6	55·8	2
	Anterior resection	2	76·0	2
	Ovarian cystectomy	1	52·0	—
	Ileocolostomy	1	81·0	—
		10 (total)	62·0 (average)	4 (40 per cent)
Non-abdominal	Thyroidectomy	1	88·0	1
	Mastectomy	2	83·5	2
	Pilonidal sinus	1	100·0	1
	Inguinal hernia	1	83·0	1
		5 (total)	87·6 (average)	5 (100 per cent)
Thoracic	Pneumonectomy	1	28·0	—
	Lobectomy	2	21·0	—
	Thoracotomy (sympathectomy)	1	37·0	—
		4 (total)	29·3 (average)	0
Thoraco-abdominal	Œsophagectomy	1	34·0	—
	Gastrotomy	1	42·0	—
		2 (total)	38·0 (average)	0

non-abdominal conditions, and upper abdominal operations show a higher incidence than lower (King, 1933; Anscombe, 1957). In this series gastric operations were made the main subject of the study, with other surgical groups established for purposes of comparison—lower abdominal, non-abdominal, thoracic, and thoraco-abdominal operations.

Two criteria were used in assessing the daily results of each patient:—

1. The maximum reduction of the forced expiratory volume as a percentage, during the first few post-operative days. The pre-operative reading was taken as 100 per cent, and the post-operative figures were given as a percentage of this value, e.g., if the pre-operative value was 4 litres and the reading on the first post-operative day was 2 litres, the post-operative reduction was given as 50 per cent.

2. The post-operative day by which the forced expiratory volume had returned to its pre-operative value. The ninth day was taken as a useful index, as it was found that in the absence of complications the patient should have regained his normal breathing capacity by then, following most abdominal operations.

The results are shown in *Table I*.

The daily readings for each patient were expressed by graph or histogram, so that the normal response to operation could be seen, and typical results are shown in *Figs.* 125–130.

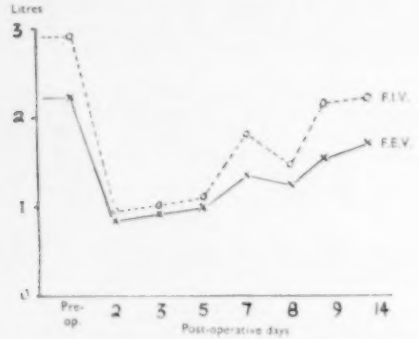

FIG. 131.—Changes in breathing capacity following lobectomy. It is shown that there is no return to normal during the first fortnight.

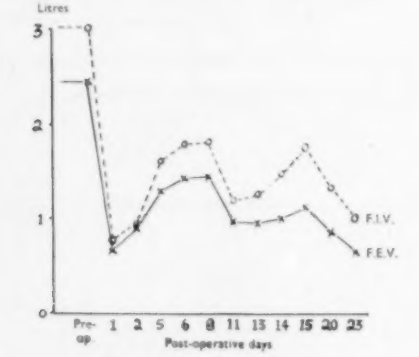

FIG. 133.—A severe reduction in breathing capacity is shown following pneumonectomy, with no improvement after three weeks.

VARIABILITY

There are many diverse factors influencing the respiratory capacity of a patient undergoing operation. All must be taken into consideration in evaluating the complete picture of response. These factors are now discussed in turn, after assessing the degree of variability of the test.

Variability of the readings was investigated in two ways—by repeating daily F.E.Vs. on the same patient pre-operatively, and by repeated F.E.Vs. of twelve or more taken at one time. These tests were carried out on normal subjects as well as on the patients in the series. It was found that the maximum value of the tracing was always the same whether three tests were done at a time or many more. It was considered, therefore, that once the test has been learnt, only three readings were necessary in order to find the maximum possible effort at that instant. When the test was repeated on different days on the same subject it was found that the percentage variation was never more than 4 per cent of the highest value, this figure being insignificant in comparison with the later changes recorded in the post-operative period.

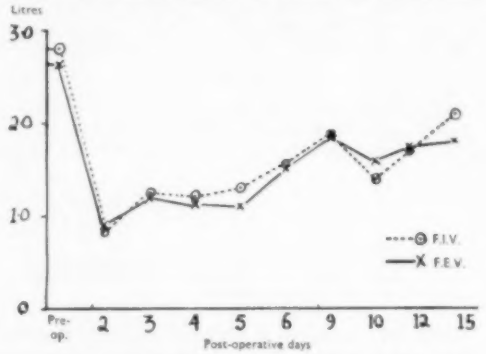

FIG. 132.—Changes in breathing capacity following œsophago-gastrectomy through a left thoraco-abdominal incision. There is no return to normal after a fortnight.

NORMAL RESPONSE TO OPERATION

It was first necessary to establish the basic pattern of alteration of post-operative respiratory capacity. This was done by analysing the cases and groups listed in *Table I*. Nearly 600 F.E.V. results were collected from the 59 cases, each result being the maximum of at least three readings. In some cases a tracing of the forced inspiratory volume was made at the end of the forced expiratory volume, thus completing the vital spirogram (Kennedy, 1953; Anscombe, 1957). However, it was found that F.I.V. changes were closely related to those of F.E.V. and there was little to be gained by adding this extra test (*Figs.* 131–133). The F.E.V. seemed a simple and adequate test of respiratory function for present purposes.

Patey (1930) found considerable fall in vital capacity after abdominal operations, with no fall after non-abdominal surgery. Pooler (1949) found

the vital capacity reduced to 50 per cent after intraperitoneal procedures, and McCleery, Zollinger, and Lenahan (1948) found the average reduction in the same group to be 53 per cent, and this order of reduction was confirmed by Powers (1928).

This investigation establishes a base line for this group of patients, but also emphasizes again the severe reduction in respiratory capacity following abdominal operations as compared to non-abdominal surgery, and especially in upper abdominal laparotomies as compared to lower abdominal operations. A small group of thoracotomies and thoraco-abdominal operations is included for comparison, and it is of

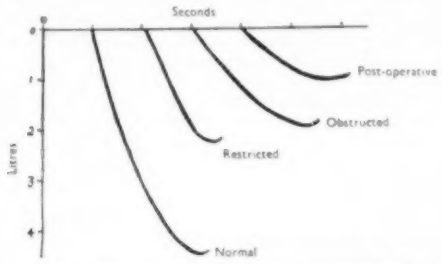

FIG. 134.—The normal F.E.V. curve compared with a restricted and an obstructed F.E.V. curve. The post-operative F.E.V. curve is a combination of restriction and obstruction.

interest to note that the upper abdominal operations approach this group in severity of effect. These results are discussed in more detail in the next section.

Many of the cases showed the maximum reduction of respiratory capacity on the first post-operative day, but others underwent a delayed maximum reduction on the second, third, or even fourth day (*Figs.* 127, 128). This can be correlated with the development of post-operative atelectasis, which reaches its maximum also between the second and fourth post-operative days, so that the first post-operative day may be a day of deceptive calm, attended by a high level of sedation and a state of euphoria which are both lost by the patient on subsequent days.

Following most major abdominal operations, the respiratory capacity had started to increase at the end of a week, and returned to normal during the second week. The histograms in *Figs.* 125–128 and graphs in *Figs.* 129, 130 illustrate this normal pattern of response. If this recovery to normal is delayed, or if there is no progress on successive days after the fourth post-operative day, then the serial F.E.V. tracings may be of diagnostic value, indicating some complication developing immediately below or above the diaphragm. In this way two complications were diagnosed, one of late basal consolidation and one subphrenic abscess, before any clinical evidence developed; the F.E.V. results showed that something was wrong, and straight radiographs then revealed whether the trouble was above or below the diaphragm.

Hugh-Jones (1958) and Thomson and Hugh-Jones (1958) have shown the different F.E.V. curves produced by a normal person, a patient with restriction of breathing (due to weakness or mechanical interference with chest wall, diaphragm, or abdominal wall movement, as in ankylosing spondylitis) and a patient with obstruction of the respiratory passages (as in asthma, bronchitis, or emphysema). A restricted F.E.V. gives the same angle with the vertical as a normal tracing, but a reduced amplitude; an obstructed F.E.V. tracing shows reduced amplitude but also a much less acute angle with the vertical which further reduces amplitude when timed. The post-operative F.E.V. shows elements of both restriction

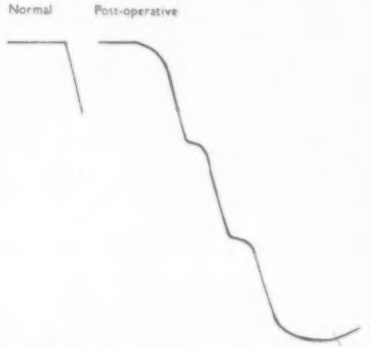

FIG. 135.—Three changes which appear on the typical post-operative F.E.V. curve: (1) The curve is flatter than normal; (2) The sharp angle of onset is lost; (3) Notching is present on the curve.

and obstruction in the tracing, with gross reduction in amplitude and flattening of the angle. A severe example is shown in *Fig.* 134. As the respiratory capacity improves towards the end of the first week, both the amplitude increases and the vertical angle becomes more acute. If either of these factors do not improve progressively at this stage then a diagnosis of residual respiratory obstruction (mucus remaining in bronchial passages, persisting bronchospasm, etc.) may be made, or of persistent respiratory restriction (such as oversedation, too tight binder, subphrenic abscess, etc.).

Two further features are characteristic of the post-operative F.E.V. and are shown in *Fig.* 135. In the normal patient the onset of forced expiration is shown by a sharp angle; post-operatively this angle is lost and becomes a curve. The same change occurs at the onset of forced inspiration. During the first few days after operation notches may be seen on the descent of the curve, and this was found to indicate retained mucus in the bronchial passages.

Two final uses of the F.E.V. in patients undergoing abdominal surgery deserve mention. In view of the post-operative changes shown, it is important to ascertain if a patient has any degree of restriction or obstruction before operation. An F.E.V. tracing is simple and quick to perform and is a useful pre-operative test in assessing any patient before abdominal surgery.

Following the performance of the F.E.V. post-operatively, many patients were enabled to cough up retained mucus. In some, carrying out an F.E.V. was almost as effective as a bronchoscopy. Thus the F.E.V. test has a diagnostic, prognostic, and therapeutic value, as well as helping in the general assessment of the pre- and post-operative patient. In elderly patients where surgery is considered, measurement

of breathing capacity is as important as a hæmoglobin estimation, and should be routine. A complex spirometer is not necessary, as testing can be done simply and easily with a peak flowmeter recording maximum expiratory flow rate.

FACTORS AFFECTING VENTILATORY CAPACITY

1. Type of Operation.—Many authors have shown that abdominal operations reduce respiratory capacity more than non-abdominal, and upper abdominal more than lower. Powers (1928) found that the vital capacity was lowered to 25–30 per cent on the first day after upper abdominal operations, to 50 per cent after lower abdominal operations, but almost not at all after operations on the limbs. Overholt (1930) described 64 per cent reduction in upper abdominal and 40 per cent reduction in lower abdominal cases on the first post-operative day. Anscombe (1957) considered the site of the incision as more important than the operation performed, and found pulmonary function reduced by 50–70 per cent with upper abdominal and by 25–35 per cent with lower abdominal incisions.

Table II.—The Effect of Vertical and Horizontal Upper Abdominal Incisions on Respiratory Capacity compared

Incision	*Vertical*	*Horizontal*
Number	29	9
Maximum reduction of post-operative F.E.V. (average)	57·4 per cent	53·8 per cent
Number of cases with F.E.V. returned to normal within 9 days	9	3

In this series the non-abdominal operations (*Table I*) showed a post-operative reduction in respiratory capacity to only 87·6 per cent of normal, and all had returned to 100 per cent within 9 days. The results showed the difference between the effects of upper and lower abdominal operations; the $F.E.V._{(1)}$ was reduced to an average of 54·6 per cent in the former and 62·0 per cent in the latter group, with 26·3 per cent in the former and 40 per cent in the latter group returning to normal in 9 days. Pulmonary resections produced the worst deterioration in respiratory capacity (reduction to 29·3 per cent) and thoraco-abdominal operations were not quite so severe in their effect (38·0 per cent); none returned to normal within 9 days. *Figs.* 131–133 illustrate these differences.

In the group of abdominal operations it does appear that the actual operative manœuvre is unrelated to the reduction in respiratory capacity; upper laparotomy has as great an effect as gastrectomy, appendicectomy as great as or greater than anterior resection. The important factor seems to be the proximity of the incision to the diaphragm.

2. Incision.—Having established that respiration is affected by the site of the incision in respect to the diaphragm, the next necessity was to find whether the type of incision affects the F.E.V. There are two main types of abdominal incision in common use—the paramedian or vertical, sometimes median, which usually does not involve cutting the muscles, and the transverse, horizontal, or oblique, which always involves muscle-cutting. Both have their advocates, but little account seems to have been taken of their effect on respiration.

In this series all the lower abdominal incisions were vertical. In the upper abdominal operations 29 were paramedian incisions and 9 were transverse or oblique. Results of these are given in *Table II*. The average maximum reduction of $F.E.V._{(1)}$ has been calculated in each group, and it can be seen that the paramedian incision group was reduced 3·6 per cent less than the transverse, on the average. By the ninth day 31 per cent of the paramedian patients and 33 per cent of the transverse had regained their normal $F.E.V._{(1)}$. Taking into account the variability of the test (*see above*), these figures are not significant. It would seem that vertical and transverse incisions have no advantage over each other in regard to post-operative respiratory capacity. Anscombe (1957) shows the difference between intra- and extraperitoneal incisions.

Table III.—Reduction of F.E.V. following Upper Abdominal Operations compared in Males and Females

Sex	*Male*	*Female*
Number of cases	27	11
Maximum reduction of post-operative F.E.V. (average)	51·0 per cent	65·0 per cent
Number of cases with F.E.V. returned to normal within 9 days	9	3

3. Sex and Age.—Holmes (1956) noted that post-operative atelectasis following abdominal surgery is commoner in males than females; respiration is mainly abdominal in men and thoracic in women. Anscombe (1957) found that men and women suffer from pulmonary complications in the ratio 2:1.

In the 38 cases following upper abdominal operations in this series, 27 were male and 11 female. The results of the F.E.V. tests in each group are given in *Table III*.

Thus males show 14 per cent more reduction in $F.E.V._{(1)}$—a significant difference. By the ninth day, 27 per cent of female cases and 33 per cent of male cases had returned to normal, but the figures are probably too small to be useful. The fact that respiratory capacity is more reduced in males than in females can be correlated with the established clinical fact that major pulmonary complications are more common in males.

The reduction in $F.E.V._{(1)}$ was compared in varying age-groups, but no relationship to age was found. Although on the whole the higher age-groups showed lower initial respiratory capacity as measured by the $F.E.V._{(1)}$, the percentage reduction following operation was no greater in the higher age-groups than in the lower. Some of the youngest of the series showed the greatest post-operative reduction, and many of the older patients showed very slight reduction in respiratory capacity.

However, the initial total respiratory reserve is less in old people than in young, and even a small reduction in old people may have more serious consequences than a large reduction in younger people. Thus, although the older age-groups do not show any greater reduction in post-operative F.E.V., yet it is a clinical fact that they have a very much higher incidence of post-operative chest complications.

4. Position.—Wade and Gilson (1951) showed that alterations in posture from the vertical to the horizontal and below had very little effect on vital capacity, but that in supine position (as compared with erect) the resting level of the diaphragm rose and the reserve air considerably decreased. However, Wilson (1927) noted that the vital capacity increased by 1 litre and the diaphragm descended 1½ in. on standing.

Table IV.—F.E.V.$_{(1)}$ Values in 8 Patients in Varying Positions

Patients	Pre-operatively					Post-operatively		
Supine	1·80	2·01	1·50	1·21	2·28	3·88	3·99	3·99
Sitting up in bed	2·02	2·38	1·72	1·22	2·38	4·20	4·12	4·46
Sitting on chair	2·19	2·38	1·66	1·36	2·44	4·46	4·28	4·64
Standing	2·32	2·43	1·90	1·42	2·45	4·41	4·41	4·70

F.E.V. tracings in this series were recorded on various patients, both pre- and post-operatively, in four positions—standing, sitting on a chair, sitting up in bed, and lying down in bed. Respiratory capacity was found to vary in the same patient in these different positions, being greatest in the standing position and least when supine. *Table IV* shows the results of tests in 8 patients, each patient being accorded a vertical column.

Thus, passing through the whole range from the supine to the standing positions, the F.E.V.$_{(1)}$ increases from values varying between 16 per cent and 27 per cent. From the supine to the sitting position increases the F.E.V.$_{(1)}$ average 15 per cent in this small group (*see Fig.* 125).

It was obviously important in view of this to test all patients in the same position, and sitting up in bed was selected as the most convenient; all readings were taken in this position. It is also important for post-operative patients to assume the erect position as soon as possible.

5. Post-operative Sedation.—During the first few post-operative days, when pain may be severe and heavy sedation and analgesia are often needed, respiratory capacity was measured immediately before and half an hour after an injection in 6 patients. The results are shown in *Table V.*

There was significant increase in 4 of the cases, showing that relief of pain can improve respiration after abdominal operation (*see Figs.* 128, 136).

6. Drainage-tube.—One factor that might be expected to influence post-operative respiration is the presence of a drainage-tube in an abdominal incision (or through a separate stab incision). Of the 38 cases undergoing upper abdominal operations, 17 were drained and 21 were not. The results of post-operative F.E.V.$_{(1)}$ changes of this group, and also of the lower abdominal group, are given in *Table VI.* It can be seen that draining the abdomen does not affect post-operative respiratory capacity.

Measurements were also made of F.E.V.$_{(1)}$ immediately before and after removing the drainage-tube post-operatively. Five patients were studied in this way and results are given in *Table VII.*

As there was significant increase in only 1 case, it follows that the presence of the drainage-tube had not caused reduction of the respiratory capacity in the majority of cases.

Table V.—F.E.V. before and after Injection of a Pain-killing Drug (Immediate Post-operative Period)

	Omnopon (gr. ½)					Pethidine (mg. 100)
Before	0·96	1·87	0·83	1·12	2·03	1·08
Half hour after	1·21	1·85	0·82	1·27	2·29	1·39
Increase	26 per cent	—	—	13 per cent	13 per cent	29 per cent

Table VI.—The Effect of Draining the Abdomen on Post-operative F.E.V.$_{(1)}$

	Drainage-tube Present		No Drain	
	Number	Maximum Reduction of F.E.V.	Number	Maximum Reduction of F.E.V.
Upper abdominal operations	17	57·1 per cent	21	54·5 per cent
Lower abdominal operations	2	76 per cent	8	58·5 per cent

In the preceding four sections the observations set down agree closely with the conclusions of other workers in the same field (Anscombe, 1957; Holmes, 1948; King, 1933).

Table VII.—F.E.V. before and after removing Drainage-tube in 5 Patients

	Partial Gastrectomy	*Partial Gastrectomy*	*Partial Gastrectomy*	*Cholecystectomy*	*Anterior Resection*
Before removal	1·66	1·49	2·05	1·62	0·73
After removal	1·62	1·90	1·60	1·52	0·71

One factor that has not received mention before is recorded in the next section and shown to have a considerable effect on respiration in the immediate post-operative period.

7. Ryle's Tube.—All the gastrectomies in the series were treated during the first few days after operation by an indwelling nasogastric tube, which was removed as soon as the aspirates became materially less than the oral intake. While the Ryle's tube was in situ, the patient would frequently complain that it hampered his coughing and breathing and often gave

rise to nausea. To assess objectively if this factor affected respiratory capacity the F.E.V.$_{(1)}$ was measured before and after withdrawal of the Ryle's tube in 8 patients and results are tabulated in *Table VIII*.

There was material increase in the respiratory capacity in 7 out of 8 cases after removing the Ryle's tube. This is of considerable importance practically. Patients will benefit in their breathing and coughing, and in certain cases pulmonary complications will be avoided, by removing the Ryle's tube at the earliest possible moment.

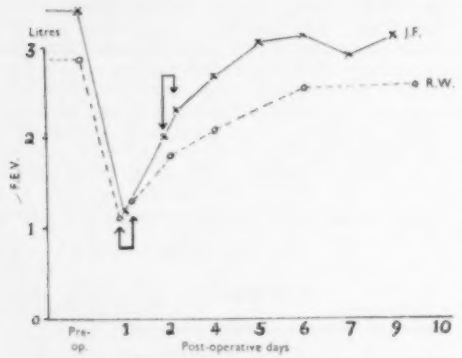

FIG. 136.—The post-operative reduction in breathing capacity of two cases undergoing partial gastrectomy where considerable pneumoperitoneum was present. These graphs can be compared with those in *Fig.* 130 and show greater post-operative reduction, although there is no delay in return to normal. The effect of injections of morphine in the post-operative periods is also shown.

In the ordinary straightforward Polya gastrectomy for duodenal ulcer it is advisable to dispense with the use of a Ryle's tube post-operatively, as it should be unnecessary from the gastric point of view, and yet its presence depresses respiration and coughing ability.

8. Increased Abdominal Tension.—Increased intra-abdominal pressure will affect respiration by raising the level of the diaphragm and curtailing its excursion, and by stretching and impeding the action of the abdominal muscles. Although thoracic movement increases in an attempt to compensate (Campbell, Howell, and Peckett, 1957), 75 per cent of ventilation in the full vital capacity results from diaphragmatic movement (Wade, 1954). The expiratory resistance is increased and the abdominal muscles, not usually used in expiration, are brought into action (Campbell, 1957). This decrease in expiratory reserve, associated with contraction of the abdominal muscles in expiration, causes a reversal of the normal pressures under the diaphragm and the intragastric pressure becomes greater in expiration than in inspiration (Campbell and Green, 1953).

Thus intestinal distension will materially reduce measurements of F.E.V.$_{(1)}$. Three examples of this reduction occurred in this series:—

1. A man aged 54 years underwent gastropexy for volvulus of the stomach. For three days after operation he developed acute dilatation of the stomach, which was suddenly relieved on the fourth day. His serial F.E.Vs. were as follows:—

Before operation: 3·17 litres.
During the gastric dilatation: 2·0 litres.
On the evening of the fourth day: 2·91 litres.

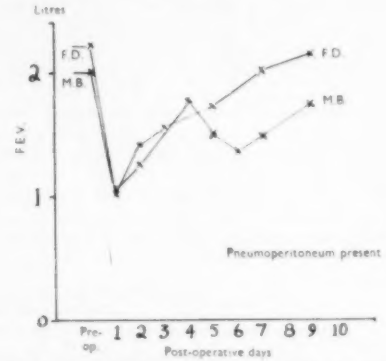

FIG. 137.—Graphs showing a reduction in breathing capacity in two cases undergoing appendicectomy, where there was considerable post-operative pneumoperitoneum. These graphs can be compared with those in *Fig.* 129 and show greater reduction, although the return to normal is not delayed.

2. A woman aged 30 years underwent gastro-enterostomy and vagotomy for duodenal ulcer and developed severe intestinal distension for four days; her bowels were opened on the fifth day. Her serial F.E.Vs. were as follows:—

Before operation: 2·5 litres.
On each of the first four post-operative days: 1·02 litres.
On the fifth day after the bowel action: 1·80 litres.

3. A man aged 31 years underwent Polya-gastrectomy for a duodenal ulcer. He developed acute retention of urine with his bladder distended up to the umbilicus on the first post-operative day, but was able to empty it spontaneously. His F.E.V.$_{(1)}$ was recorded just before and just after emptying his bladder:—

Just before: 1·05 litres.
Just after: 1·12 litres.

Table VIII.—F.E.V. in POST-OPERATIVE GASTRECTOMIES (8 CASES) BEFORE AND AFTER REMOVAL OF RYLE'S TUBE

Before	1·03	0·72	0·82	1·35	1·68	1·02	2·19	1·44
After	1·15	0·75	0·91	1·67	2·08	1·13	2·17	1·54
Increase in F.E.V.$_{(1)}$	12 per cent	4 per cent	11 per cent	24 per cent	24 per cent	11 per cent	—	7 per cent

Intestinal distension may also affect respiration in reverse, in that sudden decompression may reduce respiratory capacity. The application of an abdominal binder normally reduces the breathing capacity (Churchill and McNeil, 1927; Howkins, 1948), but in certain conditions it may assist breathing.

A woman aged 66 years was operated on to remove a huge ovarian cyst which had been causing gross intestinal distension. Post-operatively a tight binder was kept in place to prevent decompression shock. On the second post-operative day the binder was removed. The F.E.V.$_{(1)}$ was measured before and after:—

Binder in place: 1·31 litres.
Binder removed: 0·80 litres.

Presumably with long-standing intestinal distension both the abdominal musculature and the diaphragm had become stretched and could only function against resistance until recovery of tone.

Table IX.—F.E.Vs. compared in Patients with and without Post-operative Pneumoperitoneum

Group	Pneumoperitoneum	Number of Cases	Average Maximum Reduction of F.E.V.	Cases returned to Normal by Ninth Day
Upper abdominal	Absent	30	56·5 per cent	12
	Present	8	46·0 per cent	0
Lower abdominal	Absent	6	69·0 per cent	3
	Present	4	51·5 per cent	0
Lower thoraco-abdominal	Absent	1	34 per cent	—
	Present	1	42 per cent	—

9. Pneumoperitoneum.—In the 50 abdominal cases in this series 13 patients showed post-operative peritoneal air on routine post-operative radiographs—8 upper abdominal operations (all gastric operations), 4 lower abdominal (3 paramedian appendicectomies and 1 ovarian cystectomy), and 1 thoraco-abdominal (Mickulicz operation for œsophageal stricture). This group was compared with the remaining 37 cases in respect to breathing capacity, bearing in mind all the factors listed above. *Table IX* demonstrates this comparison. Examples of F.E.V. changes in cases containing air are shown in *Figs.* 136, 137 and give marked contrast to *Figs.* 129, 130.

Both upper and lower abdominal cases with air present post-operatively showed significant reduction of respiratory capacity as compared with those without air. The number returning to normal within 9 days was also greatly affected—40 per cent and 50 per cent respectively of upper and lower abdominal cases without air, but not one case with pneumoperitoneum present. Taking the gastric operations only, the 22 cases without air showed an average maximum reduction of $F.E.V._{(1)}$ of 56·7 per cent, which compared well with the larger group of upper abdominal operations in *Table IX*; all 8 cases with air present in this group were gastric operations with the $F.E.V._{(1)}$ reduced to an average of 46·0 per cent.

The effect of the air seems to be not only a greater reduction in $F.E.V._{(1)}$ but also a more prolonged reduction and delayed return to normal. In the absence of specific lung complications it was noticeable that the $F.E.V._{(1)}$ tracing followed a constant specific pattern in this group of cases with pneumoperitoneum; the vertical angle was little altered, but the amplitude was reduced and the curve became of the type associated with restricted respiration.

CONCLUSIONS

Measurement of the forced expiratory volume is a simple test which can be carried out quickly with a spirometer or even more easily with a peak flowmeter. Measurements in patients undergoing surgery show that abdominal incisions reduce greatly respiratory capacity and that upper abdominal incisions exert a greater effect than lower. The other factors depressing post-operative respiratory capacity include the presence of a nasogastric tube, the supine position, post-operative pain, abdominal distension, and the presence of pneumoperitoneum; the presence of a drainage-tube seems to make little difference and vertical incisions exert the same effect as transverse.

Acknowledgements.—I should like to thank Professor F. A. R. Stammers, for his help and advice; most of the patients were under his care. My thanks are also due to Mr. T. F. Dee, who produced the figures, and Mr. H. A. Crisp, who made the spirometer.

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RECURRENT GALL-STONE ILEUS

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RECURRENT intestinal obstruction by gall-stones is not common, but when it occurs it implies that treatment of the original obstruction has been inadequate. The characteristics of this type of obstruction are well known and will not be described here. I would emphasize, however, that symptoms of a shifting level of obstruction with intervening remissions are pathognomic of an intraluminal mass moving down the alimentary canal. It is this intermittency and subacuteness in the early stages of gall-stone ileus that leads to procrastination by both doctors and patients, and which is the most important single cause of the high mortality of the condition.

A new case is described and 11 cases have been found in the literature in which a recurrent obstruction occurred in less than one month from the first laparotomy. (*Table I.*)

Table I.—RECURRENT OBSTRUCTION FROM GALL-STONES IN LESS THAN 1 MONTH

AUTHOR	DATE	SEARCH OF BOWEL AT FIRST OPERATION	STONES FELT IN GALL-BLADDER	POST-OPERATIVE DAY OF SECOND OBSTRUCTION
1. Rogers and Carter	1958	Yes	Yes	16
2. Downes	1915	No	Not examined	2
3. Hand and Gilmore	1943	Yes	Adhesions	6
4. Macfarlane	1953	Yes	Not examined	10
5. Pybus	1922	No	Not examined	4
6. Shore, Jacob, and Cannon	1953	No	Not examined	10
7. Van Ravenswaay	1932	Yes	Not examined	10
8. Wagner	1914	Yes	Adhesions	12
9. Grey Turner	1927	No	Not examined	10
10. Foss and Dent Summers	1942	No	Not examined	5
11. Brockis and Gilbert	1957	Yes	Yes	5
12. Present case		Yes	Yes	26

Rogers and Carter (1958) recently collected 22 cases of recurrent obstructions by gall-stones, including 3 of their own; 11 recurred in less than one month, but 3 of these have been excluded: Wakefield, Vickers, and Walters's (1939), because the first obstruction was relieved without operation: Hille's (1925) because in all probability the original obstruction was caused by the second stone: Wagner's (1914) second case, because details were not given in the original literature.

CASE REPORT

Mrs. D. W., aged 49 years, was admitted to Southampton General Hospital on July 17, 1958, with 5 days' history of abdominal colic, and vomiting and constipation for 12 hours. She was not dehydrated and was very fat. She was tender all over her abdomen, with a positive release sign. Guarding was maximal in the left iliac fossa and no bowel-sounds could be heard. She was also tender in the pouch of Douglas. Her pulse-rate was 88 and her temperature normal.

On a diagnosis of peritonitis of unknown origin laparotomy was performed through a lower right paramedian incision under general anæsthesia. A gall-stone 3 cm. × 2·4 cm. was found impacted in the distal ileum, from which it was moved proximally and extracted through an ileotomy. No other stone was found in the bowel but the gall-bladder area was palpated and found to be densely adherent. Further stones could be felt, but were not removed, which proved to be a grave mistake for she could probably have withstood the further exploration necessary.

Recovery was uneventful until 26 days after the operation when she had a return of colic "just like the last time". Again there was tenderness and guarding in the left iliac fossa and bowel-sounds were obstructive. A second laparotomy was performed under general anæsthesia, this time through an upper right paramedian incision. A second gall-stone of similar size was found impacted at the site of the previous ileotomy. It was removed and again no further stones were found in the bowel. The gall-bladder was then explored and after freeing the liver from the duodenum a cavity was entered. This contained one smaller stone and represented the lumen of the fibrotic remains of the gall-bladder. The cystic duct was apparently sealed off as no bile was seen, but there was a wide fistula communicating with the first part of the duodenum. An attempt to close this with sutures and an omental patch was made and the site was drained. Apart from wound sepsis she made another uninterrupted recovery.

DISCUSSION

The poor general condition in which patients with gall-stone ileus come to laparotomy would seem to justify the emergency surgeon's maxim of "do the least necessary". However, the frequency with which more than one stone has been found in the bowel (Grey Turner, 1927, 1932; Fiddian, 1959) shows that, in all but the most desperate cases, all the small and large bowel must be examined for second stones, particularly if the obstructing stone is faceted (Hand and Gilmore, 1943; Foss and Dent Summers, 1942). That these gall-stones, large enough to block the bowel, have gained their present invidious position through a fistula connecting the gall-bladder with the alimentary canal, most commonly a cholecystoduodenal fistula, is generally accepted (Aird, 1957). Since, in the absence of pathological diverticula of the gut, it is unlikely that these stones have traversed the fistula earlier than a week or two previously, it follows that this fistula cannot yet be soundly healed and may in fact be still patent. Therefore, if there is any likelihood of a stone remaining in the gall-bladder, it

is logical to remove it too, and endeavour to close the fistula at the primary operation (Pybus, 1922; Fraser, 1954). Wright and Trafford (1953) describe a case of gall-stone ileus in which a second stone was found actually in a cholecystoduodenal fistula.

In the original accounts of only 7 of the cases in *Table I* is it specifically stated that the whole of the bowel was examined for second stones at the first operation, and it is possible that in some of the others the recurrent obstruction was caused by a stone that had already gained access to the bowel at the time of the first operation. But in the majority it is more likely that the second stone left the gall-bladder through a patent fistula in the post-operative period. In many reports the dense adhesions surrounding the gall-bladder are emphasized and have deterred surgeons from exploring this quadrant of the abdomen fully. I do not believe it is often possible to exclude the presence of further large stones under these conditions without performing a cholecystotomy. However, Fraser (1954) describes 2 cases in which small stones were felt in the gall-bladder at the primary operation, yet when interval cholecystectomy was performed in both cases the gall-bladder was found to be empty.

The sort of gall-bladder which contains massive calculi is fibrotic and no longer in communication with the common bile-duct. Moreover, the secreting mucosa is functionally destroyed and formal cholecystectomy and exploration of the common bile-duct is neither necessary nor desirable. Cholecystotomy is a fairly minor additional procedure and should be easily stood by all but the most desperately ill patients. Closure of the duodenal fistula is more difficult, but if the site is adequately drained no harm will come, and, in common with duodenal fistula after Polya gastrectomy, the discharge will cease in due course provided there is no distal obstruction. This policy of cholecystotomy in all cases of gall-stone ileus in which a large stone may remain in the gall-bladder is recommended by Brockis and Gilbert (1957), Fraser (1954), Pybus (1922), Rogers and Carter (1958) and Shore, Jacob, and Cannon (1953), but not by Aird (1957), Denneen and Broderick (1950), or Hand and Gilmore (1943). To this end, if a correct pre-operative diagnosis has been made, an upper right paramedian incision is to be recommended. This also facilitates examination of the second and third parts of the duodenum, where objects in the bowel lumen are particularly difficult to locate. If the gall-bladder is found to be un-adherent, it is my belief that the obstructing stone will prove to be a fæcolith. In one such case in which an obstructing stone was delivered from the rectum at laparotomy, eleven stones were removed from the gall-bladder, one of which was almost identical in appearance to the stone removed from the bowel. On washing them in water all eleven gall-stones sank, while the enterolith floated, and after bisection it could be seen to contain neither bile-pigment nor cholesterol.

SUMMARY

The literature on recurrent intestinal obstruction by gall-stones has been examined, and 11 cases in which the second obstruction occurred in less than a month are listed. A further personal case is described.

When operating for gall-stone ileus not only should an exhaustive search for second stones in the bowel be made, but cholecystotomy and where necessary closure of the cholecysto-entero fistula should be performed, unless the presence of residual gall-stones can be excluded or the condition of the patient will not allow it.

I am grateful to Mr. Tom Rowntree, under whose care this patient was admitted, and to Dr. Strachan, who gave anæsthetics for both laparotomies.

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TURBAN TUMOURS IN BROTHER AND SISTER

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In 1842 Henry Ancell described the case of a 52-year-old woman in whom the greater part of the face and scalp was loaded with solid tumours which varied in size from a pin's head to a horse-chestnut. The tumours first appeared when she was 14 years of age, recurred after local excision on numerous occasions, and were similar to those of her sister, mother, and grandmother. Eventually she died with intraperitoneal metastases.

Fig. 138.—*Case* 1.

This was the first description of an entity characterized by a hideous appearance, rapid recurrence after excision, distinctive histology, and familial incidence. There have been nearly as many different names for the condition as there have been cases described, and it is probably rather rare, for Evans in 1954 mentioned 47 cases in addition to his 2 personally studied patients, and it is doubtful if many escaped his notice.

CASE REPORTS

Case 1.—A. S. (45283.59), male, aged 61 years. Railway guard. When 40 years old he first noticed small lumps on his head, which steadily grew bigger. He was referred by his own doctor to the skin department, whence Dr. Pegum kindly referred him to this unit. Both ears had been injured in the first world war, and both mastoids had been operated on in 1932, leaving him partially deaf.

On examination his scalp was covered by countless small nodules interspersed amongst others varying in size from 1 in. to 2 in. in diameter (*Fig.* 138). The smaller ones were sessile but others were pedunculated. The largest tumours were to be found on his scalp, but similar nodules were scattered over the face, nose, and ears, and there were others over the trunk and neck. Apart from a well-marked thoracic kyphoscoliosis and bilateral conductive deafness there was no other abnormality. Blood-pressure 150/100. Chest radiograph normal.

On April 22, 1959, a number of the more prominent lesions of the scalp, chest, and shoulders were removed, with a considerable improvement in his appearance.

Further excisions of tumours from the scalp were undertaken on Aug. 19, with a satisfactory result. There was no significant change in his appearance when he was seen on Dec. 8. He continues to be seen at regular intervals in the out-patient clinic.

Case 2 (59617.)—Mrs. L. R., aged 58 years. Housewife. Ever since she was 48 years of age she had noticed several lumps on her head, which had gradually enlarged. At times they stung, and when combing her hair they occasionally bled or exuded a whitish material. She persistently refused to take medical advice, and to hide her deformity she lived with her head covered. The improvement in her brother's appearance led her to attend the out-patient clinic with him.

On examination there were innumerable red, hairless, painless tumours all over her scalp, varying in size from a pea to a tangerine; some were soft, others hard; some were pale, others red or cyanosed. The general appearance defies description (*Figs.* 139, 140). A kyphoscoliosis rather similar to that of her brother and moderate deafness were the only other findings of note. Her deafness was considered to be due to some wax in her external meatus rather than to the tumours overlying the external ear. On Dec. 11, 1959, ten of the larger tumours, and on Dec. 15 another dozen, were removed. Recovery was uneventful. Her personality changed considerably as a result of the comparative improvement in her appearance, and she has been seen at regular intervals since then. On each occasion we have tried to persuade her to have the remaining tumours removed, but she politely refuses.

Further inquiry revealed that the mother of these two patients had had several operations for lumps on her head, and the maternal grandfather and an aunt both had similar nodules about the head. Other members of the family were said to be unaffected (*see Fig.* 141). Although none of the children of *Case* 2 have developed these tumours, they are probably as yet too young.

DISCUSSION

When Ancell (1842) showed the first case his colleagues suggested so many different names for it that he felt obliged to confine himself to a description of the case "with two accurate drawings hoping it will prove acceptable as a contribution towards a more complete history of diseases called malignant".

Subsequent authors have not kept to this prudent policy, and the following names have been suggested for the same condition—syphonoma, tomato tumour, hydradenoma, nævo-epithelioma adenoides, epithelioma adenoides cysticum, non-papillary hyalinizing hydradenoma, multiple familial benign nodular intra-epidermal basal-cell epithelioma, endothelioma capitis of Kaposi, Spiegler's tumour, cylindroma—but never Ancell's tumour. Many of these terms are unsatisfactory as they suggest an origin from sweat glands which has not been conclusively established. We

favour the use of descriptive or eponymous names and prefer to follow the current usage of the term 'turban tumour' to designate that group of skin tumours showing a predilection for the scalp and face. Other typical cylindromatous neoplasm (Type 1, *Fig.* 142). The other (Type 2, *Fig.* 143) was an adenoid type of growth which was present in only *Case* 1, where it was closely associated with the cylindromata. It was

FIG. 139.—*Case* 2.

FIG. 140.—*Case* 2.

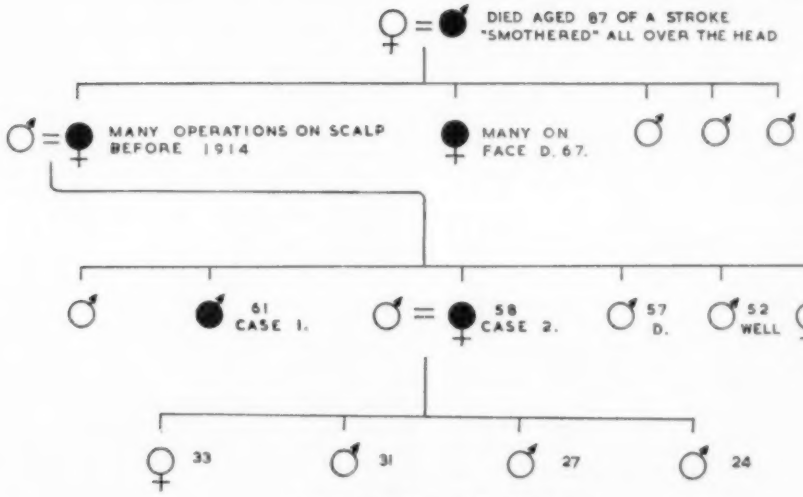

FIG. 141.—Family tree of the present cases.

terms must be reserved for the histologically different subgroups, the most frequent being the cylindroma, but epithelioma adenoides cysticum is also encountered.

Histology.—In the first case there were two histological types of tumour: the predominant type was a seen in none of the material from the second case, and has been described before in a case of turban tumour by Savatard (1938). Both types showed occasional tenuous connexions with the epidermis stretched over them, but no connexions with any recognizable appendages of the skin could be seen

in serial sections. Where present the sweat and pilosebaceous glands were normal. Both tumours have a similar morphology, and were made up of intertwined, coiled, and ramifying cylindrical masses of tumour cells, but differed in the amount of stroma between these tumour masses.

Type 1 (*Fig.* 142).—The cylindromatous tumours were composed of close-packed masses of cells of

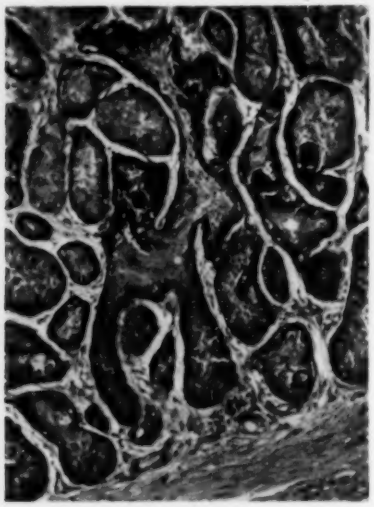

FIG. 142.—Typical cylindromatous neoplasm as seen in *Cases* 1 and 2. P.A.S. (× 105.)

basal type, with a peripheral palisade pattern. The outer or basal ends of the cells rested on a prominent, thick, hyaline membrane. In the middle of the tumour masses were collections of similar hyaline material, sometimes connected with the peripheral hyaline membrane, suggesting in serial sections that in most instances they arose as infoldings of the peripheral membrane. None of the hyaline material was found within cells.

There were occasional duct-like spaces lined by one or two layers of flattened cells in parts of the tumour.

Type 2 (*Fig.* 143).—The less common adenomatous tumour was made up of basal cells which were rather larger and less hæmotoxaphilic than in Type 1: there was a peripheral palisade, but no limiting hyaline membrane. Instead, the border was wavy, and strands of stromal collagen were incorporated deeply into the clumps of tumour. In these incorporated strands of stroma there was every gradation of change from the normal, through a nodular hyalinized form, to a frankly mucinous cyst—a typical feature of this adenoid tumour. There was never any intracellular mucin or hyaline, but the walls of the tumour cells were not always well defined, particularly near the cysts. Abundant loose collagenous stroma separated the clumps of tumour.

A variety of different methods were used to stain the material from these two cases (*Table I*). They show that the hyaline material from Type 1 behaves as a mesodermal scleroprotein, somewhere between reticulin and collagen. It contains hyaluronic acid, and a mucopolysaccharide ground substance which is insensitive to hyalase. The hyaline material lying within the tumour masses, though not quite so rich in tryptophane, has the same staining properties as that on the periphery.

In Type 2 the stroma is less sensitive to hyalase, and where it has undergone mucinous transformation,

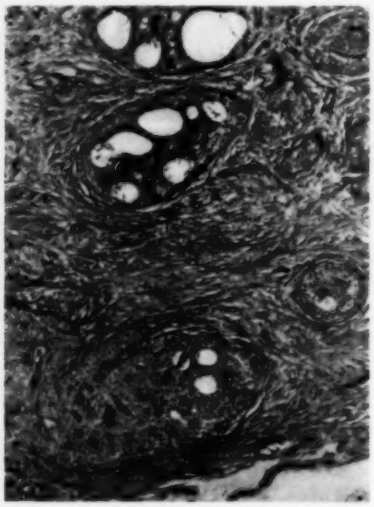

FIG. 143.—Adenomatous neoplasm seen only in *Case* 1. P.A.S. (× 97·5.)

a mucopolysaccharide, free from hyaluronic acid, has been either unmasked or taken up. There is as well a hyaluronic acid containing mucin. That there are also free phosphate radicles is suggested by the orange colour obtained with methyl-green-pyronin, and the golden hue with the von Kossa.

Previous authors have discussed some of the properties of the hyaline material in Type 1 tumours: Evans (1954) found that it stained pink with P.A.S. but did not stain like collagen with Van Gieson. Braun-Falco confirmed this (1955), demonstrated that tyrosine was present, and concluded that the hyaline was a polymerized glycoprotein of epithelial origin. On the contrary, our observations tend to show that it is of mesodermal origin. This was also suggested by Sutherland (1956).

Turban tumours have been thought to arise either from one or other of the epidermal appendages, or from undifferentiated rests (Gates, Warren, and Warvi, 1943). Recent text-books favour an origin from sweat-glands (Lever, 1954; Allen, 1957; Lund, 1957) or hair matrix (Ormsby and Montgomery, 1954). Our own observations do not permit any dogmatic statement about the origin of these tumours: the cylindromatous Type 1, whilst lacking glycogen, has no features which positively show an origin either in pilosebaceous follicles or in sweat glands. The progressive change in the stroma of the adenoid Type 2 and its incorporation into the epithelial masses is similar to the change seen in the maturation of the hair bulb (Montagna, 1956) and might

suggest an origin in this part of the pilosebaceous follicle. Although Sutherland (1956) showed connexions between the tumour and pilosebaceous follicles, he none the less favoured an origin from sweat-glands and regarded these connexions as secondary. Had connexions been observed between the tumour and sweat-glands, his explanation would be more acceptable.

Malignancy.—Most authorities state that turban tumours are benign; but there are few where the follow-up has been long enough to exclude ultimate malignant change. In Luger's case (1949) the cylindromatous turban tumour had been subjected both to surgery and to eight years of continuous irradiation, which may have precipitated the malignant change and the metastases. The metastasizing tumour in Lausecker's second case (1952) appears to have been an epithelioma adenoides cysticum type of turban tumour. We may perhaps disregard Ancell's original case for want of adequate histology, but until long-term results are known it would be wise to regard these lesions as potentially malignant.

Genetic Factors.—Many reports emphasize the familial incidence of these tumours. Good family pedigrees have been published by Savatard (1938), Luger (1949), Chalstrey (1955), and Regan (1956). In both familial and sporadic cases women are more frequently affected than men; Evans (1954) found 30 females compared to 17 males, but Ronchese (1940) had a female preponderance of 4 in his 44 cases and in Luger's family more males were affected than females. While there is therefore reason to doubt a sex linkage in the inheritance, the condition does appear to be inherited as a dominant factor. It must be remembered that two-thirds of the recorded cases give no familial history (Evans, 1954).

Because of the genetic background, the characteristic area of involvement, and the general benignity of these tumours, they are evidently nævoid and not a variant of a basal-cell carcinoma as suggested by Willis (1953).

Symptoms.—In nearly every case the tumours are first noticed after puberty, suggesting that hormones play a part in their aetiology (Evans, 1954). At first miliary, when fully grown the tumours may be as big as oranges, and though sometimes solitary (Sutherland, 1956) they are usually very numerous, covering the scalp, face, or ears, and sometimes hiding the eyes (Gabarro and Baker, 1945), with outlying groups of tumours on the neck, trunk, genitalia, or groins.

There may be spontaneous pain: Ancell's original patient said that just before it rained the lumps would "smart and leap" a good deal; Evans's patient was troubled by intense irritation at night; our own *Case* 2 complained of occasional stinging in the swellings. More often, however, the tumours only hurt if they are accidentally pinched or injured. Sometimes the patients have been deaf—usually from tumours in the ears.

Without any doubt the worst symptom is the deformity. This, and the difficulty of keeping the head clean, may turn the patient into a solitary recluse (Gabarro and Baker, 1945).

Differential Diagnosis.—There are few conditions which can look quite like a turban tumour. The two chief histological types have been discussed already. Metastases from a distant primary carcinoma rarely appear upon the scalp, but, when they do, they may so closely imitate the turban tumour as to deceive even the most experienced (Ronchese, 1940).

Table I.—Summary of Staining Methods

Stain	Cylindroma		Adenoid		
	Basal	Intratumoral	Stromal	Intratumoral	Cyst
Van Gieson	Orange-pink		Yellow, or few fine red fibrils	Pale pink	Pale pink
Reticulin	+	+	+	+	+
Anisotrophy	−	−	A few anisotropic fibres	−	−
Congo red	− or ±	± or −	± or −	− or ±	−
Sudan black	Thin layer	adjacent to cells	±	±	−
P.A.S.	+ + +	+ + +	+	+ + +	+ + +
P.A.S. after diastase	+ + +	+ + +	+	+ + +	+ + +
P.A.S. after hyalase (Benger)	+ + +	+ + +	+	+ + +	+ + + or ±
Thionin (metachromasia)	−	−	−	−	+
Thionin after hyalase (Benger)	−	−	−	−	−
Thionin after ribonuclease (Armour)	−	−	−	−	±
Alcian blue	+	+	±	± or +	+ + +
Performic acid, alcian blue (Adams and Sloper, 1956)	±	±	− or ±	− or ±	+ + +
Alcian blue (saline control to hyalase)	+ +	+ +	+	+	+ + +
Alcian blue after hyalase (Benger)	±	+	±	±	+ + +
Methyl green, pyronin (pyroninophilia)	−	−	−	−	Orange
Methyl green after ribonuclease (Armour)	−	−	−	−	−
Von Kossa	−	−	−	−	− or gold-brown
Millon	+	−	+	−	−
p-Dimethylaminobenzaldehyde-nitrite for tryptophane (Adams, 1957)	+	−	+	+	−
Thyoglycolate-ferric-ferricyanide for disulphide groups (Adams, 1956)	+	+	± or −	±	+

Treatment.—In the belief that most of these tumours follow a benign course, local excision has usually been the first line of treatment, and will sometimes suffice. It is not always easy—the tumours are highly vascular, and special precautions to cut down blood-loss at the time of operation are essential. Preliminary ligature of the main vascular supply of the scalp has been attempted (Gabarro and Baker, 1945), and we have found local infiltration with adrenaline 1–100,000 solution of great assistance. Pre-operative preparation of the scalp, no matter how careful, always leaves nooks and crannies amongst these close-packed pedunculated tumours, which may not be properly sterilized, so that infection is an additional hazard.

Early recurrence is the rule. The only way the patient can be rid of his tumours is to resect all the tumour-bearing skin. Indeed, the growths may be so extensive that this is the only practicable measure. It has been necessary to remove the entire scalp, with both ears, and the skin from most of the nose (Gabarro and Baker, 1945; Evans, 1954; Regan, 1956). The raw area is covered with split skin, and the patient supplied with a suitable wig.

Radiotherapy has been tried by Luger (1949) and Evans (1954), who was able by its means to destroy small growths entirely, but larger ones underwent a temporary and patchy necrosis, only to come back as big as ever in a few weeks. When one examines the skin between the more obvious tumours, it is so riddled with further small nodules that to destroy these would require total destruction of the scalp.

In the two cases described here the initial plan has been local excision of the tumours in stages. Recurrences are highly likely, and it may then be necessary to remove the scalp: both patients are being carefully followed up, with this, and the ultimate possibility of a malignant change, in mind.

SUMMARY

A brother and sister both with turban tumours are presented.

Certain histological and histochemical features are described and discussed with relation to the nomenclature and possible histogenesis of these tumours.

The treatment and management of patients with these disfiguring tumours is described.

We wish to thank Professors V. W. Dix and D. S. Russell for their helpful advice and criticism; Mr. J. King and Mr. P. Broadberry for their photographic assistance; and Miss G. Moules for help with the manuscript.

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ANGIOSARCOMA (HÆMANGIOBLASTOMA) OF THE BREAST

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THIS case is reported first because of the rarity of unequivocal malignant tumours of blood-vessels in general and of those arising in the breast in particular, and secondly because it seems worthwhile to re-stress the fairly constant clinico-pathological pattern of this deadly disease.

CASE REPORT

The patient was aged 27 years and of central European origin. In September, 1956, she had noticed a small soft lump in the right breast. This was treated conservatively in her own country for fourteen months, and in November, 1957, it was removed. Histology was said to show a benign hæmangioma, but the sections were not available for study. The tumour recurred almost immediately, and in 1958 the patient came to England.

EXAMINATION showed considerable enlargement of the right breast and in one area a bluish colour was seen through the skin. The skin temperature was raised over this area and several soft, compressible, lobulated masses were palpable. These were quite painless and no bruit was heard with a stethoscope. There were no enlarged lymph-nodes in the axilla or neck, the opposite breast was normal, and a general examination revealed no further abnormalities.

An arteriogram was performed by direct puncture of the left subclavian artery with a tourniquet tied around the middle third of the upper arm; 15 c.c. of 60 per cent urografin were injected. The vessels were clearly seen and in the capillary phase pooling of contrast medium was observed in the breast superficial to the pectoral fascia (*Fig.* 144). The pools varied in size from mere venous dilatation to large cavernous spaces. This seems a useful

investigation which, as far as the writer is aware, has not previously been described.

Removal of the lump was carried out by the excision of the upper two-thirds of the breast, with preservation of the nipple and areola. The lower third was left behind and appeared macroscopically normal. The lesion is shown in *Fig.* 145.

HISTOLOGY showed numerous blood spaces of cavernous type lying within the breast substance. These

FIG. 144.—Pooling of contrast medium in the right breast.

FIG. 145.—Operation specimen showing main tumour.

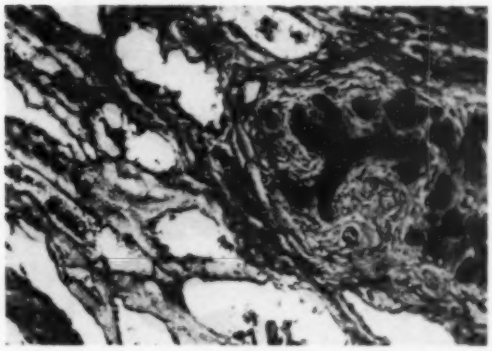

FIG. 146.—Cavernous spaces within breast substance. H. and E. (× 55.)

appeared quite benign (*Fig.* 146). Other areas, however, showed marked endothelial proliferation resulting in areas of solid tumour with mitotic figures (*Figs.* 147, 148). A diagnosis of angiosarcoma was made. Six weeks later the patient noticed a small discoloured area in the affected breast. This was excised and showed blood-filled spaces of benign type. Five months after removal of the main tumour a plastic operation was performed, and a nodule was found deep to the scar. Histology showed proliferation of endothelium suggestive of malignancy. Accordingly, a radical mastectomy was performed and a focus of angiosarcoma was found invading pectoralis minor (*Fig.* 149). The axillary lymph-nodes were not invaded. Three months later the patient complained of severe pain in the back and radiography showed marked destruction of D.9, with collapse and compression. During the ensuing weeks further metastases appeared in rapid succession. These were situated in the cervical vertebræ, left humerus, right acetabulum, and right lung, and there were multiple subcutaneous deposits, particularly in the regions of the face and scalp. These were treated with radiotherapy and chemotherapy and the problem of treatment is discussed later. The patient died approximately 3½ years after the first appearance of the lesion.

DISCUSSION

Great caution is necessary before the diagnosis of angiosarcoma is made. Highly vascular sarcomas and carcinomas, hamartomatous lesion, hæmangiopericytomas, and mesenchymomas with vasoformative areas must be excluded. Willis (1953) has stressed that genuine malignant tumours of vascular origin are very uncommon and in the breast they are exceedingly rare. The scarcity of cases makes a precise estimate of incidence impossible. Enticknap (1946) suggests an incidence of 0·03 per cent of mammary neoplasms, and with the cases of McClanahan and Hogg (1954) the incidence was 1 in 1700. Stewart (1950) suggested an incidence of 1 angiosarcoma for every 2000 breast carcinomas. Hill and Stout (1942), using the term 'malignant hæmangio-endothelioma' described three types which have been observed in the breast:—

Type 1: The primary lesion and the metastases look histologically benign. Such lesions have been called 'benign metastasizing hæmangiomas' and a multicentric origin may be difficult to disprove.

Type 2: The primary lesion looks innocent but definite malignant appearances are seen in the metastases.

Type 3: The tumour is histologically malignant from the outset.

Such a subdivision has only a limited value but it does stress the likelihood of the histology of any such tumour looking deceptively innocent. The importance of a thorough examination of multiple sections cannot be overemphasized, and, even if no aggressive features are noted, there is no guarantee that benign behaviour will parallel the histology.

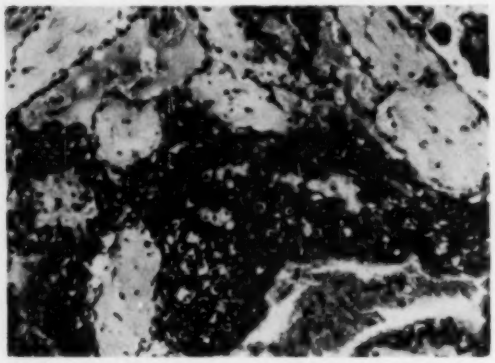

FIG. 147.—Cavernous spaces and endothelial proliferation. H. and E. (× 75.)

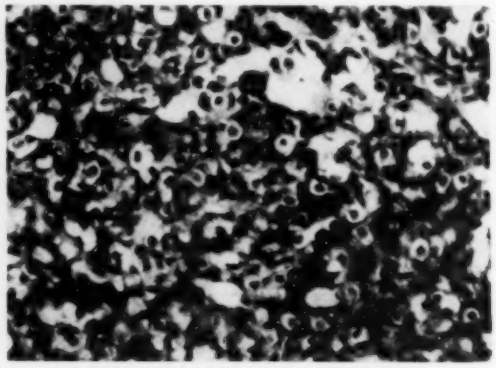

FIG. 148.—High-power view of cellular areas. H. and E. (× 175.)

Table I shows the fully documented and acceptable cases. Certain other cases have been mentioned in the literature but have been omitted because of inadequate documentation. These cases include those of Onsy (1931), Melville and Bloodgood (1933), Sherry (1934), Geschickter (1945), and Mallory (1949). It seems certain, however, that the disease, though

Table I.

CASE	AUTHOR	AGE	ORIGINAL HISTOLOGICAL ASSESSMENT	TREATMENT	COURSE OF DISEASE
1	Batchelor (1959)	25	Malignant	Simple mastectomy	Survived 6 months; died with subcutaneous nodules and probably liver metastases
2	Borrmann (1907)	26	Benign	Local excisions	Survived 21 months; died with multiple metastases
3	Enticknap (1946)	20	Malignant	Simple mastectomy	Survived 9 months; died with pulmonary metastases
4	Ewing (1940)	?	Benign	None described	Survival time unknown. Multiple metastases in skin, mucous membranes, and lungs
5	Hill and Stout (1942)	65	Malignant	Radical mastectomy	Survived 2 months; died with multiple metastases
6	Hill and Stout	19	Benign	Injection with quinine HCl and ethyl carbonate. Simple mastectomy	Survived 17 months; died with multiple metastases
7	McClanahan and Hogg (1954)	22	Malignant	Local excision; radium needles, radical mastectomy, and radiotherapy	Survived 24 months; died with pulmonary metastases
8	McClanahan and Hogg	18	Benign	Local excision. Radiotherapy to metastases	Survived 42 months; died with multiple metastases
9	McClanahan and Hogg	29	Malignant	Simple mastectomy	Survived 14 months; died with multiple metastases
10	McClanahan and Hogg	23	Benign	Local excision. Simple mastectomy	Survived 66 months; died with multiple metastases
11	McClanahan and Hogg	28	Malignant	Radiotherapy followed by radical mastectomy	Survived 22 months; died with pulmonary metastases
12	McClanahan and Hogg	23	Malignant	Radical mastectomy	Survived 16 months; died with multiple metastases
13	Patrick, Jarvie, and Miln (1957)	30	Benign	Simple mastectomy removal of axillary contents. Deep X-ray therapy; no details given	Survived 18 months; died with multiple metastases
14	Patrick, Jarvie, and Miln	23	Malignant	Simple mastectomy	Survived 26 months; died with multiple metastases
15	Patrick, Jarvie, and Miln	18	Malignant	Simple mastectomy	Survived 10 months; died with multiple metastases
16	Pulford (1925)	38	Malignant	Local excisions	Survived 36 months; died with multiple metastases
17	Robinson and Castleman (1936)	18	Benign	Local excision. Radiotherapy to metastases	Survived 42 months; died with multiple metastases
18	Shore (1957)	31	Benign	Local excision. Radiotherapy. Simple mastectomy	Survived 25 months; died with multiple metastases
19	Stout (1943)	68	Malignant	Simple mastectomy	Survived 3 months; died with multiple metastases
20	Tibbs (1952)	37	Benign	Simple mastectomy	Survived 24 months; died with multiple metastases

rare, is commoner than the figures in *Table I* would suggest.

A study of the literature and *Table I* shows that the disease has a fairly constant natural history. The following are the main features:—

1. All the cases have occurred in women.

2. It is predominantly a disease of young women —average age 29 years.

FIG. 149.—Slices of breast from radical mastectomy specimen, showing muscle invasion.

3. Considerable enlargement of the breast is common.

4. In nearly 45 per cent of cases the initial histology has suggested a benign lesion.

5. The longest survival was 5½ years, but the average time was only about 2 years.

6. Dissemination is by the blood-stream and, while metastases are widespread, they show a particular predilection for the lungs, skin, subcutaneous tissues, and bones.

Problems of Treatment.—It is clear from *Table I* that no effective treatment has so far been devised. While the short survival times undoubtedly reflect the malignant nature of this disease, a contributing factor may have been a failure to institute prompt and radical treatment at an early date. At times this delay has been the result of misinterpretation of the apparently innocent histological picture which is often seen. In the present case the primary lesion was treated surgically but the patient had had the lump fourteen months before arrival in England. Radiotherapy was given to the metastatic lesions as follows:

From the Van de Graafe 2 MeV generator, the affected dorsal vertebra received 5060 r (skin dose) over a period of 6 weeks, while 1840 r were given to the cervical vertebra over the same period of time. A metastasis near the right clavicle was treated with 15-curie telecobalt 60 to 4095 r in 3½ weeks, while the upper end of the humerus received 3848 r in 3 weeks. The subcutaneous deposits were treated with 80 and 250 kV X-rays over 8 weeks. There was a definite response to this treatment, marked by disappearance of some of the subcutaneous deposits and recalcification of the bony lesion with healing of a pathological fracture. Thus radiotherapy, particularly of megavoltage type, may be more effective than has been previously supposed. The patient also received 25 mg. of Durabolin intramuscularly weekly for eight weeks.

It is clear that prompt diagnosis, immediate radical surgery, plus pre-operative and or post-operative radiotherapy, offer the best chance of improving the prognosis in these cases.

SUMMARY

1. A case of angiosarcoma of the breast is described.

2. Problems of histology and treatment are discussed.

I am indebted to Sir Stanford Cade for permission to publish this case and for his comments and to the Department of Medical Photography, Westminster Hospital.

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MASSIVE UNILATERAL HYDRONEPHROSIS IN EARLY INFANCY

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Massive unilateral hydronephrosis producing noticeable enlargement of the abdomen at any age is uncommon: at birth it is a rarity. It is usually due to obstruction at the pelvi-ureteric junction, but published case reports do not always distinguish between this type of hydronephrosis and that due to causes lower in the urinary tract associated with a hydroureter.

In the middle of the eighteenth century Glass (1747) described a young woman who died at the age of 22 years as a result of a gigantic right hydronephrosis, not above a pint less than 30 gall. wine measure (109·5 l.) of fluid being obtained at autopsy. The left kidney was normal. According to her mother, the patient was 'born dropsical' with an enlarged abdomen, but never had any swelling of the thighs or legs.

Johnstone and Browne (1921) gave an account of a female infant who was apparently normal at birth, but who became ill after 4 hours with coldness and blue extremities. The infant suddenly became white and died 2 hours later. At post-mortem examination a right hydronephrosis the size of a cricket-ball was found, and the left kidney, which seemed small, showed dilatation of the pelvis and calices.

A boy, aged 8 years, who was born with an unusually large abdomen, was reported by Hahn (1924). A few months before admission the abdomen began to enlarge alarmingly. At operation there was a right hydronephrosis containing 'about 1 gall. (4 l.) of fluid'. The right ureter could not be found.

McCrea (1930) reported a case of a very large hydronephrosis in a male infant which first produced distension of the abdomen at the age of 3½ months. This distension increased and a left nephrectomy was carried out at the age of 4 months, the specimen removed weighing 1 lb. 4 oz. (567 g.). The obstruction was apparently at the pelvi-ureteric junction.

Fig. 130.—*Case* 1. Intravenous pyelogram (and key drawing) at the age of 6 weeks, before operation.

The right kidney was palpated at operation and was presumed to be normal. Subsequent progress was satisfactory.

A more severe example was that described by Rosedale (1935). He reported a female infant, whose abdomen was noted at birth to be distended by a palpable, fluctuant mass in the right upper quadrant. The mass became progressively larger and a clinical diagnosis of congenital polycystic kidney was suspected. The infant died on the eighteenth day of life. At post-mortem examination a large hydronephrosis was found, the lower three-fourths of the ureter being cord-like and solid. The left kidney and ureter were normal.

Among Kretschmer's (1937) own series of 101 examples of hydronephrosis, occurring in children up to the age of 13 years, were 9 infants under the age of 6 months, the youngest being 3½ weeks old. One of his cases, who had a left nephrectomy carried out at the age of 7 months, was first noticed by the mother to have swelling of the abdomen when 2 months old and this had become progressively larger.

White and Wyatt (1942) studied a series of 30 children with symptoms due to an aberrant renal vessel. The youngest to be operated on was aged 3 months, another being aged 10 months.

Nineteen cases of pelvi-ureteric obstruction in children under 14 years of age were reported by Soley (1946), and his youngest patient was aged 8 months.

Campbell (1951) published a series of 512 clinical cases of hydronephrosis in childhood and in just

over one-quarter of these the obstruction was at the pelvi-ureteric junction or adjacent ureter. In the vast majority of this smaller group the condition was unilateral. His whole clinical series included 21 male and 40 female infants up to the age of 6 months.

Gross (1953) investigated 145 children with hydronephrosis due to pelvi-ureteric obstruction and the youngest of these was aged 1 month.

Nixon's (1953) series comprised 77 patients with treated hydronephrosis, who were seen at the Hospital for Sick Children, London, from January, 1929, to August, 1950. This included only 1 case under the age of 6 months (Bearn, 1956). Comparison may be made with the 39 children with hydronephrosis from the Royal Victoria Infirmary, Newcastle, who were encountered during the years 1943–52 and were reported by Turner (1954). The 2 youngest infants in this latter series were aged 4 weeks and 3 months respectively, but in both instances the obstruction was at the bladder neck or in the posterior urethra.

Bearn (1956) described 2 infants with congenital pelvi-ureteric hydronephrosis. In each the abdomen had been enlarged since birth. The first infant was operated on at the age of 3 months and a plastic repair performed on the right side. The hydronephrosis was bilateral and a similar operation was carried out on the other kidney 19 days later. The second infant underwent operation at the age of 6 weeks, when an enormous right hydronephrotic kidney was removed. As a subsequent pyelogram showed hydronephrosis also on the left side, this kidney was subjected to a successful plastic repair at the age of 1 year.

The kidneys produce urine as early as the fourth month of fœtal life (Kjellberg and Rudhe, 1949), so that if there is obstruction at the pelvi-ureteric junction hydronephrosis may be present at birth. If the

FIG. 151.—*Case* 1. Intravenous pyelogram (and key) at the age of 16 months.

condition affects both kidneys and is severe the infant is usually stillborn. On the other hand, in rare instances the infant is born alive with an enormous unilateral hydronephrosis causing at birth a noticeably enlarged abdomen which may produce an increasing degree of embarrassment to respiration and to feeding. Two such cases are now reported, which were operated on at the age of 7 weeks and 1 day respectively.

CASE REPORTS

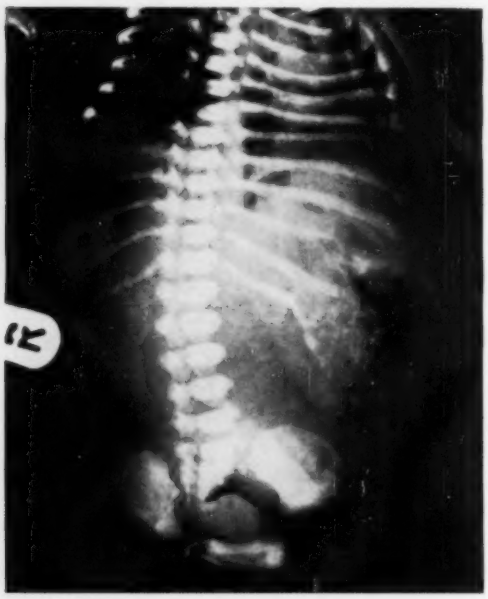
Case 1.—R. P. This boy was born at home by normal delivery and weighed 8 lb. 4 oz. (3·7 kg.). He was seen at birth to have a large abdomen which progressively increased in size. Vomiting began at the age of 1 month and persisted intermittently; because of this, the child was referred to hospital at the age of 6 weeks.

ON EXAMINATION.—The abdomen was distended and tense, there being a prominent network of fine veins over the abdominal wall. The maximal abdominal girth was 18 in. (45·7 cm.). On palpation, a very large smooth mass could be felt occupying the whole of the right side of the abdomen and passing across the midline; the lower edge could be felt just above the pubic symphysis. Rectal examination clearly demonstrated its lower pole. It could be readily transilluminated and the effect was spectacular.

A pyelogram, using hypaque intravenously, showed good concentration of dye in the left kidney with slight pelvic distension but no recognizable excretion on the right side (*Fig.* 150). Some dye appeared in the lower third of the right ureter and this was thought to be due to reflux.

The blood-urea was 58 mg. per 100 ml. The urine contained no albumin and was microscopically normal.

AT OPERATION.—After 6 days in hospital the infant was submitted to surgery, an intravenous infusion of N/5 sodium chloride with 4·3 per cent glucose having been started. Under general anæsthesia, through a loin



incision, a tense, retroperitoneal cystic mass was encountered which was found to be a hydronephrosis. Four hundred and forty ml. fluid were aspirated initially so that the mass could more easily be delivered. Once this was done, there was little difficulty in separating the very thin wall from the peritoneum and surrounding tissues. The right suprarenal gland was found plastered over its upper pole and was gently dissected free. The right renal artery and the vein were ligated with catgut and the ureter ligated 1 in. below the pelvi-ureteric junction. The total fluid content of the hydronephrotic sac removed was 25 oz. (711 ml.). The wound was closed without drainage and the child was given 70 ml. blood intravenously. He made an uninterrupted recovery, being discharged from hospital 11 days after the operation.

PATHOLOGY.—The specimen was devoid of any obvious renal tissue but on microscopic section slight fibrosis and round-celled infiltration with dilatation of some collecting tubules were detected in the capsule of the cyst.

PROGRESS.—When last seen at the age of 16 months, the child was thriving. The urine contained no albumin, was microscopically entirely clear and produced no growth on culture. The blood-urea was 39 mg. per 100 ml. Intravenous pyelography was then repeated and the left kidney showed good function, but again with evidence of early hydronephrosis (*Fig.* 151), perhaps a little more advanced than it had been 14 months earlier.

Case 2.—L. R. This girl was born at home by normal delivery with a birth-weight of 8 lb. (3·6 kg.) and was noticed at birth to have a very large abdomen.

When examined on the same day, the abdomen was markedly distended, particularly over the right upper two-thirds. This was due to a mass which was dull on percussion and extended round into the right loin posteriorly. The mass was cystic in nature but could not be transilluminated.

A pyelogram was carried out on the day after birth, using hypaque intravenously. This confirmed the presence of a huge mass occupying the whole of the right side of the abdomen and displacing the bowel to the left. The left kidney and ureter were normal but no recognizable excretion of dye could be seen in the right kidney (*Fig.* 152). On the same day catheterization produced 5 ml. dark urine, containing no organisms and only 3

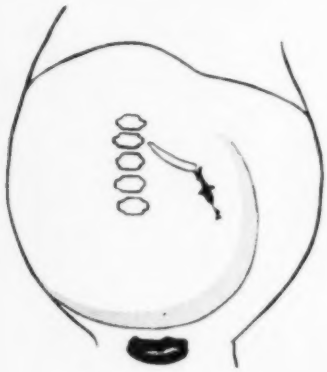


FIG. 152.—*Case* 2. Intravenous pyelogram (and key) at the age of 1 day, before operation.

leucocytes per c.mm. The blood-urea was 64 mg. per 100 ml.

AT OPERATION.—In view of the very gross abdominal distension, operation was carried out on the day after birth. An enormous hydronephrosis was exposed through a right loin incision. Once again fluid was aspirated in order to deliver the cystic swelling out of the wound. On this occasion, however, dissection of the wall from the peritoneum anteriorly and from the suprarenal above was tedious and difficult in spite of there being no apparent preceding infection.

When the right suprarenal gland was dissected clear, the renal vessels and ureter were ligated and the mass was removed. The wound was closed without drainage and 70 ml. blood were given intravenously. A total of 19 oz. (540 ml.) urine was obtained from the hydronephrotic sac.

Post-operative progress was uneventful and the baby went home 9 days after the operation. The weight on discharge was 7 lb. 2 oz. (3·2 kg.).

PROGRESS.—When last seen in the out-patient clinic at the age of 12 months, the infant was thriving. The urine contained no albumin and 1 leucocyte per c.mm. microscopically. The blood-urea was 36 mg. per 100 ml. Intravenous pyelography again showed an entirely normal left kidney (*Fig.* 153).

DISCUSSION

At birth the gut is still devoid of air so that a large collection of fluid or a mass of considerable size is necessary to produce any noticeable degree of abdominal distension immediately after delivery. Of the solid renal swellings, a Wilms' tumour must be

suspected in early childhood. It rarely, however, attains large proportions in the neonatal period.

Cystic renal swellings may be due to congenital unilateral multicystic kidney disease: there are at least 46 recorded cases (Moe and Crofford, 1960). These may present in the neonatal period and early operation may be carried out, as in the infant recorded

by Fillis, Grantham, and Cullen (1958) which was operated on when 15 days old. In such instances the outline of the mass is sometimes irregular on palpation, whereas in cases due to hydronephrosis the outline is smooth.

Longino and Martin (1958) analysed a series of 32 infants admitted to the Children's Hospital in Boston because of abdominal masses noted during the first day of life. Of the 17 proved to be of renal origin, 1 was a case of Wilms' tumour. Thirteen were due to a unilateral multicystic kidney and 2 to bilateral multicystic kidneys. Only 1 was found to have hydronephrosis. Other causes found in this series included a large hepatic solitary cyst and an enormous malignant hepatoma.

Small infants rarely show sufficient abdominal relaxation to permit detailed palpation, but examination may be rewarding if carried out during a feed. If the fluid is under tension, the swelling may be surprisingly firm to the touch. Transillumination may give a useful indication of the fluid nature of the swelling, as in our first case. In the second case, however, it was not successful, presumably because of a thicker, more adherent wall. With very large swellings fluctuation may be confirmed on rectal examination.

It would seem imperative to carry out intravenous pyelography before operation in all similar cases, even as an emergency procedure. In the newborn infant 10 ml. hypaque should be given intravenously to ensure good visualization of the renal calices. We have previously encountered an infant with a moderately severe right hydronephrosis in whom pyelography revealed a severely hypoplastic left kidney, so that if operative removal is contemplated, the state of the contralateral kidney must be known beforehand. Palpation alone at the time of operation can be misleading.

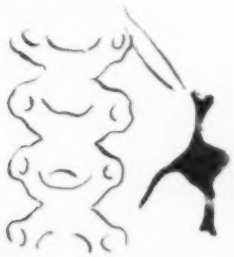


FIG. 153.—*Case* 2. Intravenous pyelogram (and key) at the age of 12 months.

While plastic reconstruction operations have been performed even on small infants with hydronephrosis (Bearn, 1956), complete removal of the sac was the only possible treatment in our two cases. When the sac is of great size, careful and extremely tedious dissection may be called for. Blood-loss must be restored.

Although in each case the remaining kidney was still functioning well one year after operation, it will be of interest to observe whether progressive hydronephrosis develops or not. We do not know of the long-term follow-up of other published cases.

SUMMARY

Two infants are reported who were noticed at birth to have considerable abdominal swelling due to a massive unilateral hydronephrosis. Operative removal was carried out at the age of 7 weeks and 1 day respectively with complete relief of symptoms.

A review of the literature suggests that congenital hydronephrosis presenting in this way is a rarity. The diagnosis and management are discussed.

Acknowledgements.—We wish to thank Miss B. M. Becket, medical artist, and Mr. E. Stride, A.I.B.P., medical photographer, for their help in preparing the illustrations.

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OCCLUSIVE DISEASE OF THE AORTA AND ITS MAIN BRANCHES

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THE problem of occlusive disease of the aortic arch and its branches has interested surgeons rather intensively in recent years. Although the first information regarding this interesting syndrome was given as early as 1875 by a British author (Broadbent, 1875), the Japanese claim some originality in its description and many have associated the name of Takayashu, the Japanese ophthalmologist, with this syndrome. Takayashu (1908) in reality described only the peripapillary flush in ocular manifestations of this disease and did not deal with the aortic occlusion. Broadbent's description of the condition in 1875 was so clear that it is worthwhile reproducing part of it here.

"On further examination, the innominate was found not only to be narrowed at its origin, but rigid. An atheromatous patch surrounded the mouth of the vessel involving the structures both of the aorta and the innominate; it was hard and brittle without being distinctly calcareous and separated readily from the other tunic. Immediately above its origin, the innominate enlarged to its usual size and possessed its normal elasticity. Here then was the explanation of the absence of pulse with a full vessel at the right wrist."

However, no other English, European, or American authors seemed to be interested in the condition for many years. A number of case reports appeared in the Japanese literature mainly from the ophthalmologists (Sato, 1928; Oota, 1940), and a very detailed description appeared from the pen of Shimuzu and Sano (1951). This article contained the catchy phrase "pulseless disease" and came to the general knowledge of many workers because it was abstracted extensively in the *Journal of the American Medical Association* of the same year. In 1954, Ask-Upmark, from Scandinavia, reviewed all the cases reported in the world literature, excluding Japan. The total number of cases reported outside Japan was 28 at this time and this included 2 of his own. In 1957, Kalamansohn and Kalamansohn made a further review of all cases reported, totalling 90, of which 5 were in the United States. They presented the sixth case in the series. In England, isolated cases had been intermittently described during the years (Skipper and Flint, 1952; Lewis and Stokes, 1942).

That the syndrome is really not so uncommon has been evident since 1957. A spate of reports concerning the clinical, diagnostic, and the therapeutic problems associated with the disease have emanated from the North American continent and to a lesser extent from other countries. Also many cases have been effectively treated by surgical methods (De Bakey, Morris, Jordan, and Cooley, 1958; De Bakey, Crawfoord, Cooley, and Morris, 1959; Bahnson, Spencer, and Quattlebaum, 1959; Rob and Wheeler, 1957). A limited number of cases have also been reported from India (Shikhare, 1921; Sengupta and Ghosh, 1957; Pahwa, Pandey, and Gupta, 1959). However, although much is now known about the clinical, and to a lesser extent about the therapeutic, aspects of the disease, its aetiogenesis and pathology remain largely obscure. Its exact nomenclature has not been properly settled. As will be evident later on in this presentation, the name 'pulseless disease' is not correct as many cases have palpable pulses. Takayashu's disease, or as others have called it, 'Martorelli's syndrome', are eponymous terms, but can be objected to, because the disease really differs markedly from the limited description available from the writings of these authors. In fact, one of the objects of the present study would be to show that this disease does not always conform to a particular clinical pattern, that the pathological process may involve diverse, distant, and unrelated vessels, and that although the disease is usually and mainly confined to the aortic system, it will be shown that the pulmonary vascular tree is not immune. Such being the case, the non-committal term 'occlusive disease' has been used in the present study and it is hoped with more information about the pathologic process involved a better generic name would emerge in future.

Material.—Between November, 1953, and June, 1960, 10 cases of occlusive disease of the aorta and other vessels were seen by us. The clinical or the laboratory data are not exhaustive in all the cases, but all of them fall in the group of so-called 'occlusive disease' and have therefore been included in the present study.

CASE REPORTS

Case 1.—Female, 13 years, admitted on Nov. 24, 1953. Well and active until 11, when for a year she suffered from irregular fever and sore throat. Tonsils were removed. Later she complained of breathlessness

on accustomed exertion. She also complained of weakness, palpitation, coldness in legs and feet, and warmth in the upper extremities. These gradually progressed and she was confined to bed.

On Examination.—Patient was stunted and anæmic. No cyanosis, jaundice, or œdema. Blood-pressure: R.A. 250/140 mm. Hg and L.A. 245/145 mm. Hg. No B.P. in legs. Pulsation absent in abdominal aorta, femorals, popliteals, or tibials.

Heart: Apical impulse 5th left intercostal space ¾ in. outside mid-clavicular line. It was heaving in character.

Fig. 154.—Shows narrowing and thickening of wall of aorta commencing (A) 4·5 cm. from the left subclavian artery. (*By courtesy of the 'American Heart Journal'.*)

A localized loud systolic murmur was heard over left second intercostal space 1 in. from the sternum. The aortic second sound was ringing and a soft systolic murmur was present in this area. A systolic murmur was present in the back at the interscapular region.

Ophthalmoscopic examination showed blurring of disk margins, retinal arteriosclerosis and superficial hæmorrhages and exudates.

Investigation.—Hæmatology—Normal. Wassermann reaction—Negative. Radiography—No rib notching. E.C.G.—Left ventricular strain.

At Operation (Dec. 24, 1953).—Left thoracotomy. Starting at mid-thoracic region and extending to the diaphragm, the descending aorta was constricted and thickened. The mediastinal pleura was firmly adherent to this segment of aorta. The case was deemed unsuitable for repair at this time.

The patient died 7 days after operation. Partial autopsy was allowed. Coarctation of the thoracic aorta began 4·5 cm. beyond the origin of the left subclavian artery and extended to the level of the diaphragm measuring 8·5 cm. (*Fig.* 154). The thickness of the wall and the size of the lumen of the left common carotid and subclavian arteries at their origin and of the aorta at the level of the diaphragm may be seen in *Fig.* 155 A, B, C, respectively. On microscopic examination, the constricted portion of the aorta showed thickening of the intima and early atheromatous changes in it. There was perivascular round cell infiltration in media and adventitia.

Case 2.—Female, aged 15 years, was admitted in another hospital in April, 1955, with history of dyspnœa on exertion, palpitation, headache, weakness of 2 years' duration. Her weakness, particularly in the legs, progressively increased until she was admitted to the hospital.

On Examination.—Slightly built, anæmic girl. Blood-pressure: R.A. 220/110 and L.A. 215/112. B.P. not recordable in the lower extremity.

The pulses in the upper arm were forcible but were not palpable in the legs. Abdominal pulsation very feeble. Femoral and popliteal pulsation not palpable.

Investigations: Radiography—No rib notching. Angiocardiography—The coarctation was not properly visualized. E.C.G.—Left ventricular strain pattern.

At Operation (May 24, 1955).—Left thoracotomy. An exact replica of the findings as in the previous case was present. The descending thoracic aorta from about 1 in. below the left subclavian to the diaphragm was thickened, indurated, and somewhat constricted. The pleura over this area was adherent and thickened. The thorax was closed. This patient died 36 hours after the operation. Autopsy was refused.

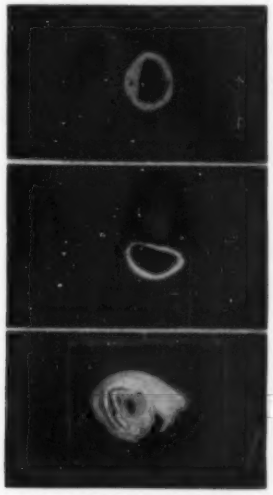

Fig. 155.—Shows thickness of the wall and the size of the lumen of (A) left common carotid, (B) left subclavian, and (C) thoracic aorta at the level of the diaphragm. (*By courtesy of the 'American Heart Journal'.*)

Case 3.—Female, 17 years, average built woman, complained of palpitation, dyspnœa, headache, and precordial pain of about 6 months. There was no definite history of rheumatic fever but she did complain of occasional joint pains particularly after exertion.

On Examination.—Blood-pressure: R.A. 180/100 mm. Hg and L.A. 180/95 mm. Hg. Pressure not recordable in the inferior extremity. Upper arm and carotid pulses full. Abdominal aorta pulsation feeble. Femoral and popliteal very faint.

Heart: Enlarged. Apex beat 6th intercostal space, ½ in. lateral to middle line and heaving in character.

Systolic murmur all over precordium, also heard at the interscapular region in the back.

Investigations: Urine—S.G. 1006. Alb. + +. E.C.G. —"Bundle branch block with evidence of degenerative changes in the heart and myocardiac weakness." Fundal examination—No significant change. Aortography through left brachial artery showed an area of block in upper abdominal aorta proximal to renal arteries with poststenotic dilatation (*Fig.* 156). Significant pressure gradient was obtained at this level.

Following aortography, the patient began to complain of acute abdominal pain frequently and 3 days later died suddenly. Autopsy was performed.

Autopsy Report.—The abdominal aorta below the level of the renal arteries was thin, elastic, and had a smooth surface. A marked thickening of the abdominal aorta starts just above the origin of the renal arteries—the maximum reaching about 7 cm. above the level where there is a constriction in the lumen. There is narrowing of the lumen of the aorta with thickened walls of

about 2·5 cm. (*Fig.* 157). All the coats showed thickening, the intima being intensely thickened, with a ragged surface, and it could be easily separated from the overlying muscle layer. Above the area of maximum narrowing of the lumen the diameter of the lumen gradually increased till the aorta pierced the diaphragm.

HISTOLOGY REPORT.—Above the coarctation, the aorta showed thickening of all the coats. There was fibrous

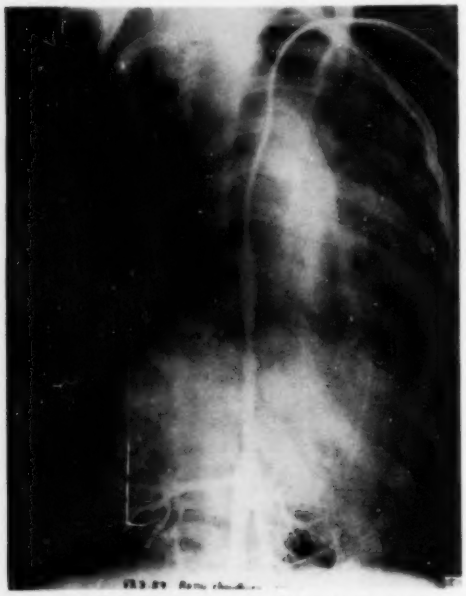

FIG. 156.—Shows constriction of the upper abdominal aorta proximal to the renal arteries with an area of post-stenotic dilatation.

thickening of intima with focal collection of lipoid-laden macrophages. In the deeper layers of this fibrous thickening numerous newly-formed elastic fibres are seen (fibroelastosis). The most characteristic features of the medial change were disruption and disorganization of the elastica and increase of PAS positive, metachromatic substance in the medial coat. There are small empty spaces between the muscle-fibres along with patchy vacuolation within the fibres themselves.

At the site of coarctation, the changes were morphologically similar to that seen above. The thickening of the intima was, however, more marked and there was increase of periadventitial connective tissue with focal perivascular infiltration of lymphocytes.

Below the coarctation, the wall of the aorta appeared normal except for slight fibrous thickening of the intimal coat as also some lipoid-laden macrophages.

The cœliac artery showed atheromatous thickening of the intima. The pulmonary artery showed subintimal atheroma.

Case 4.—Female, aged 20 years, admitted on Sept. 14, 1959. Dyspnœa, palpitation, weakness, and occasional giddiness of two years' duration. This patient was first seen in 1955 in the neurology department with symptoms simulating intracranial tumour. This was later diagnosed to be due to intracranial hæmorrhage due to ruptured berry aneurysm.

ON EXAMINATION.—Pulse 84. Respiration 20. Blood-pressure: R.A. 220/60 and L.A. 220/60. R.L. 145/50 and L.L. 140/40.

AUSCULTATION: Systolic murmur over aortic area conduction towards neck. Systolic murmur at the back in the interscapular region.

Radiography.—(1) L.V. hypertrophy. (2) Doubtful rib notching right 5th rib. (3) Patchy calcification over arch and descending thoracic aorta (*Fig.* 158).

Aortography: Catheter through brachial artery got obstructed in arch at origin of left subclavian artery.

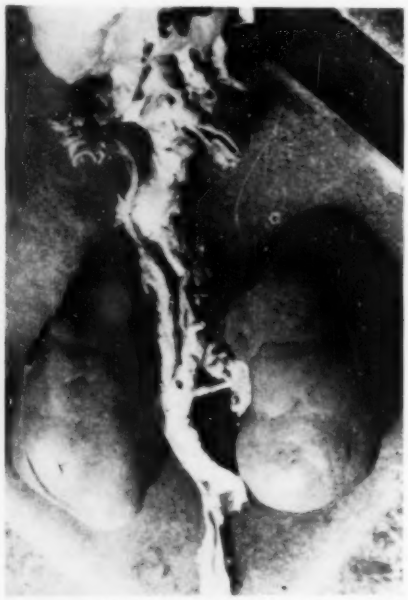

FIG. 157.—Shows marked thickening of abdominal aorta proximal to the renal arteries. The orifices of the abdominal aortic branches are surrounded by indurated areas and are somewhat constricted.

1ST OPERATION (Dec. 1, 1959.)—Exploration through left thoracotomy. A segment of aorta 3 in. long beyond left subclavian artery was dilated and there was massive calcification in the wall of this involved segment. The area was hard and non-compressible. The aortic wall here was hard as rock and unindentable (*Fig.* 159). The subclavian artery was dilated and its wall near the origin of the vessel was also slightly affected. Thoracotomy closed.

2ND OPERATION (Jan. 2, 1960).—After left thoracotomy, the previous findings were confirmed and a suitably sized knitted dacron graft was put in place between the subclavian artery and the descending thoracic aorta to bypass the involved segment of descending thoracic aorta. Biopsies were taken.

HISTOLOGY.—(1) Diffuse marked thickening of intima. (2) Narrowing of media with area of scarring and disorganization. (3) Adventitia showed marked thickening. (4) Vasa vasorum showed elastic hyperplastic thickening.

Case 5.—Female, aged 10 years, complained of multiple joint pains, low rise of temperature, dyspnœa, and palpitation for 8 months.

ON EXAMINATION.—Pulses present in upper extremity but impalpable in the lower extremity. Blood-pressure: R.A. 200/120 mm. Hg and L.A. 205/110 mm. Hg. No pressure in lower limbs.

Heart: Enlargement of left ventricle. Grade I systolic murmur over precordium. E.C.G.—Nothing significant.

Pre-operative transventricular aortogram—Narrowing of lumen of descending thoracic aorta just beyond left subclavian artery.

1ST OPERATION (Dec. 18, 1959).—Left thoracotomy. The pleura over the arch and the descending thoracic aorta were firmly adherent. The subclavian and the internal mammary arteries were dilated. The descending aorta was narrow and thickened and at places bossy on the surface. An area of marked constriction was seen 2 in. above diaphragm. Aortography showed diffuse obstruction in the aorta. Chest was closed.

FIG. 158.—Shows calcification over the aortic arch and upper part of descending thoracic aorta.

FIG. 160.—Picture taken at operation showing constricted segment of thoracic aorta involving an area of about 2½ in.

2ND OPERATION (April 19, 1961).—An area of about 2½ in. of the middle segment of the thoracic aorta was found constricted (*Fig.* 160). Repeat aortography on the table revealed that this segment did not fill at all. The segment below the area showed post-stenotic dilatation (*Fig.* 161). The abdominal aorta was visualized by a second injection below the area of constriction. This showed that the abdominal aorta was patent and there was filling of the renal and the mesenteric blood-vessels (*Fig.* 162). The iliac vessels were extremely thin and attenuated. Incision of the aorta above the constricted segment showed that there was marked endarteritis, while the wall of the aorta below the constriction was markedly hypoplastic. By-pass graft using knitted dacron tube (De Bakey) was instituted between the proximal and distal segments of the descending thoracic aorta (*Fig.* 163). It was not considered advisable to resect the involved segment.

Case 6.—Female, aged 19 years, complained of weakness, giddiness, and progressive dimness of vision of 3 years' duration. By the time she was admitted to the hospital in June, 1955, she had lost vision in both eyes. She also developed weakness in the right upper and lower extremities and ultimately developed hemiplegia.

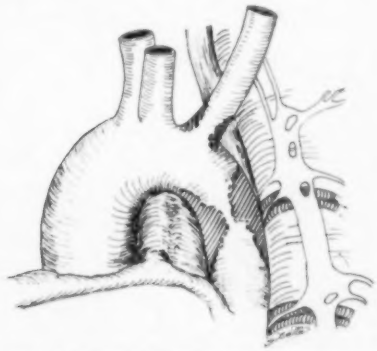

FIG. 159.—Line drawing showing area of involvement of the aortic arch and the descending thoracic aorta.

FIG. 161.—Aortographic study at operation showing area of constriction and post-stenotic dilatation.

Pulses were not palpable in either superior extremity. They were palpable in both inferior extremities. Carotid pulses very feeble. Blood-pressure: Upper extremity, not recordable. Lower extremity, 125/90 mm. Hg.

ON EXAMINATION.—

Heart: Systolic murmur over base of heart.

Ophthalmoscopy: Venous congestion and atheromatous patches.

INVESTIGATION.—A catheter in the left brachial artery was taken up to near the aortic arch and an aortographic

FIG. 162.—Abdominal aortography showing patency. The iliac vessels are extremely attenuated.

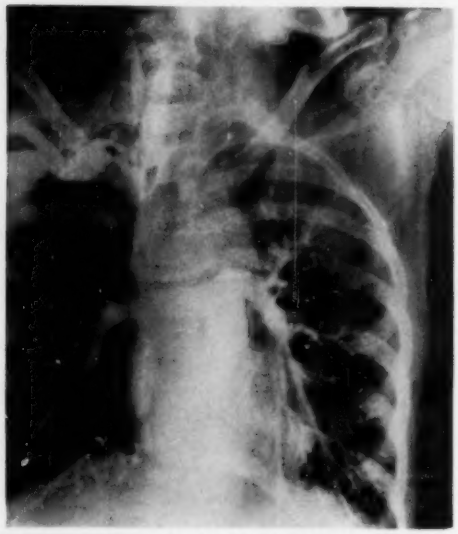

FIG. 164.—Pulmonary angiography showing non-visualization of the upper branch of the right pulmonary artery.

study was attempted. There was complete block of the left subclavian and the dye did not enter the aorta. There were numerous collaterals.

Surgery was not advised in this case.

Case 7.—Male, aged 21 years. Headache, giddiness, and blurring of vision for 1 year. Complained of sudden onset of headache and blood-pressure recorded at this time was 220/110 mm. Hg. He was admitted and was discharged with the diagnosis of migraine.

Second admission, February 22, 1960.

ON EXAMINATION.—Blood-pressure: R.A. 130/60 mm. Hg and L.A.—not recordable. R.L. 160/70 mm. Hg and L.L. 160/80 mm. Hg. Pulses: very feeble on the left brachial and radial. Left carotid pulse also feeble. Other pulses normal. There was an area below the inferior angle of the left scapula where there was peripheral arterial pulsation.

Heart: Aortic area.

There was harsh systolic murmur. This was conducted to the neck. The systolic murmur was also heard at the back in the interscapular region.

Ophthalmoscopy: No significant change.

E.C.G.: Left ventricular preponderance.

FIG. 163.—Picture taken at operation showing a dacron graft in place by passing the area of constriction in the thoracic aorta.

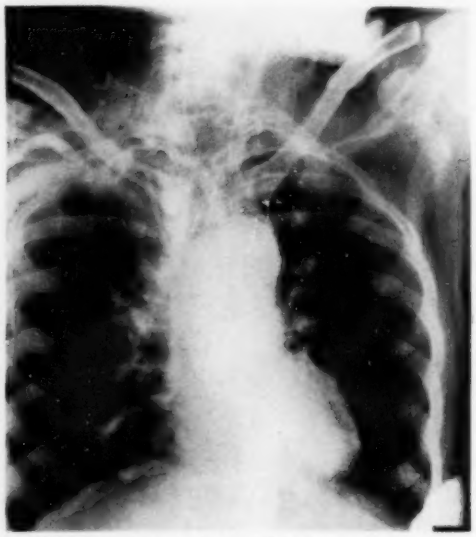

FIG. 165.—Poor visualization of the left common carotid, the left subclavian arteries, and constriction of the root of the innominate artery.

Angiography: (1) Non-visualization of the upper branch of right pulmonary artery (*Fig.* 164). (2) Non-visualization of the left common carotid artery and the left subclavian artery (*Fig.* 165). (3) Constriction of the innominate artery.

The patient did not agree to operation.

Case 8.—Female, aged 28 years. Pain in back, headache, and weakness 16 months following intermittent rise of temperature lasting for a month.

ON EXAMINATION.—
Pulse: (1) Pulses in the upper extremity not palpable. (2) Carotid pulses feeble.

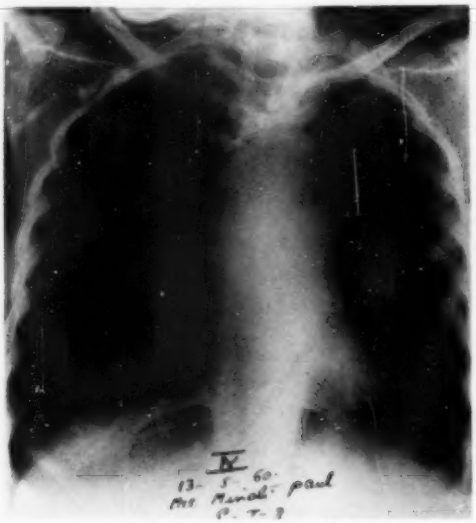

FIG. 166.—Aortography showing non-visualization of the innominate and the left subclavian arteries, and poor visualization of the left common carotid artery.

FIG. 167.—Aortography showing non-visualization of left subclavian artery and loss of continuity of a segment of descending thoracic aorta below the left subclavian artery.

FIG. 168.—Aortography showing non-visualization of the left common carotid artery and visualization only of the root of the left common carotid artery. Note good visualization of the left vertebral artery. Note also constriction of the descending thoracic aorta just distal to the left subclavian artery with an area of post-stenotic dilatation.

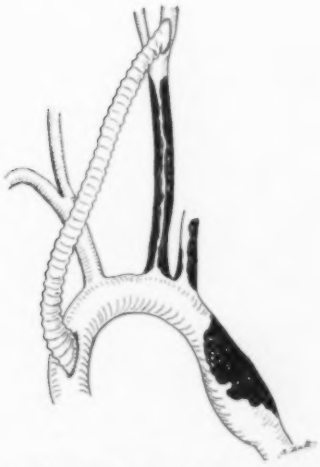

FIG. 169.—Line drawing showing the long dacron graft between the ascending aorta and the bifurcation of the left common carotid artery.

First admission in hospital 8 months back when pulse and blood-pressure were recorded in the upper extremity. Had 2 fainting attacks, the second one a few days before her second admission to hospital on May 6, 1960.

Blood-pressure: R.A. not recordable and L.A. not recordable. R.L. 130/60 mm. Hg and L.L. 110/60 mm. Hg.
Heart: Apex beat 6th space 1½ in. away from midclavicular line. L.V. enlarged. Systolic murmur over

the base conducted upwards. Systolic murmur also heard in paravertebral region.

E.C.G.: Normal.

Ophthalmoscopic Examination: Normal.

Angiocardiography (*Fig.* 166): (1) Huge dilatation of ascending aorta. (2) Innominate and left subclavian arteries not visualized. (3) Left common carotid was faintly visualized.

Arterial Biopsy (Intercostal artery): (1) Narrowing of lumen. (2) Marked medial hypertrophied focal area of degeneration in the hypertrophic muscle-cells. (3) No intimal thickening. (4) Absence of arteritis or pan-arteritis.

Case 9.—Male, aged 28 years, was admitted in a tuberculosis sanatorium with pulmonary tuberculosis involving left lower lobe. Had repeated hæmoptysis. Sputum was positive. On bronchography, the left lower lobe was found atelectatic and bronchiectatic. There was history of headache for 2 years prior to detection of pulmonary tuberculosis.

On Examination.—

Pulses: Right upper extremity, normal. Left upper extremity, not palpable. Right carotid pulsation, normal. Left carotid pulsation, feeble.

Blood-pressure: R.A. 140/70 mm. Hg. L.A. not recordable.

Heart: Systolic murmur at base of heart; also heard at the back interscapular region.

Angiography (*Fig.* 167): (1) Innominate, right subclavian and right common carotid arteries visualized well. Left common carotid artery also seen. (2) Left subclavian not visualized. (3) Loss of continuity of aorta below the left subclavian artery.

At Operation (May 24, 1960).—Left thoracotomy as for left lower lobectomy. Moderate narrowing of the descending thoracic aorta 1½ in. below the left subclavian artery affecting an area of about 2 in. Thrill over the coarcted area. Left subclavian artery non-pulsatile and cord line. Lumen nearly completely occluded. The left subclavian artery was opened; there was flow in fine stream. Biopsy was taken.

Histology.—"Main lesion is seen in media. (1) Wide areas replaced by hyaline structure which take intense P.A.S. stain (mucopolysaccharide). Scattered in the area irregularly are intensely hæmato-oxophylic (chromatotrophic) substance which is not calcium. There is marked elastosis associated with areas of fragmentation and irregular clumping. (2) There is excess of P.A.S. positive material in the adventitia along with elastosis. (3) The intima is thickened. The essential characteristic is medial degeneration with excess of mucopolysaccharide, chromatotrophic substance, and abnormal elastosis."

Case 10.—Male, aged 35 years. Three months prior to admission in hospital had sudden attack of fainting followed by right-sided hemiplegia. He gradually recovered from this partially. On admission, there was weakness in right upper and lower extremities and the patient walked with a limp. There was also exaggerated knee jerk and the plantar reflex was extensor.

On Examination.—Pulsation, very feeble in left carotid, axillary, and brachial vessels. Right side normal. Blood-pressure: R.A. 150/80 mm. Hg and L.A. 90/70 mm. Hg. R.L. 130/84 mm. Hg and L.L. 130/84 mm. Hg. Systolic murmur over the aortic area and in the root of the neck.

Angiography (*Fig.* 168): Showed that the left common carotid and the left subclavian were faintly visualized. There was also an area of constriction in the descending aorta just distal to the left subclavian with post-stenotic dilatation.

At Operation, June 23, 1960.—By an incision in the neck, the whole length of the left common carotid and its bifurcation were exposed. The common carotid felt solid and tough. Very weak pulsation was seen at the bifurcation. The ascending aorta was exposed by splitting the sternum, and a dacron graft put in between the ascending aorta and the bifurcation of the common carotid (*Fig.* 169). The wall of the aorta was friable and held stitches badly and occasioned considerable loss of blood. Biopsy was taken from left common carotid artery.

Histology Report.—Sections from different parts of the artery have been serially studied. (1) Apparently occluded segment. (2) Segment below and above. (3) Apparently normal segment. (4) Marginal areas.

1. Segmental atherosclerotic lesion with a recanalized organized thrombus. The segmental lesion is very characteristic with vascularization of the thickened intimal and subintimal connective tissue and fragmentation of the medial elastic coat.

2. Sections above this area show recanalized thrombus, but the segmental intimal lesion is less marked.

3. The normal portion also shows atherosclerotic intimal thickening and the extent of this thickening progressively becomes more (4) at the marginal area. In all the segments the vasa vasorum is fairly thickened with focal collection of small round cells round them.

The adventitia in the affected segment is markedly thickened with focal collections of inflammatory cells.

DISCUSSION

If one is to judge the incidence of occlusive diseases of the aorta and its branches by cases hitherto reported in the literature, one would think that they are rare. However, the fact that we have had 10 cases during the course of the last 7 years, and 4 of them within the last 6 months, makes us feel that they are really not very uncommon and, if one is interested and is on the look-out, cases are liable to turn up fairly frequently. It is evident that many cases are missed at the early stages and that unless dramatic complications ensue, such as hemiplegia or blindness, or unless persistent or recurrent symptoms such as headache, giddiness, weakness, etc., continue to trouble the patient, they do not come to the attention of physicians or surgeons and are not properly investigated or diagnosed.

This is illustrated significantly in 2 of our cases. In one, the patient was diagnosed at the first admission in hospital to be suffering from migraine and was treated on this line; the second case was also treated at her first admission as a case of pyrexia of non-specific origin. Pulses were said to be present and normal in upper extremities in both cases at this time. Later, the dramatic onset of syncope with absence of radial pulsation in one case, and the persistence of major symptoms in the other case, caused them to be further investigated, and the diagnosis could be confirmed. It appears to us that these cases, in their variegated clinical patterns, will increasingly come to the attention of clinicians in future.

By strict definition, the so-called 'pulseless' disease refers to cases involving the roots of the innominate, the left common carotid, and the left subclavian arteries. It will be seen that in this series we have included cases affecting other parts of the aorta and even other arteries. There is a good deal of justification for this. It is hard to imagine why an occlusive arteritis process should pick out only the mouths or the trunks of the head vessels of the aorta and leave other branches and other portions of the aorta untouched. It will be seen also that of the 6 cases of occlusion of various portions of the

descending thoracic aorta in our series, there were at least 3 cases where the left subclavian artery and other head vessels were also affected *pari passu*, proving thereby that the occlusive process may have many patterns.

Occlusion of the descending thoracic aorta has been described in the literature as a separate clinical entity. Dubost and Blondeau (1957) described 2 cases of their own, and collected 15 other cases from the literature. In 1955 we also reported one similar case as one of diffuse coarctation (Konar, Chowdhuri, and Basu, 1955). Recently Flynn and Kattus (1959) reported a case affecting the middle segment of the descending thoracic aorta, where, on exploration, there was calcification on the wall and massive end-arteritis in the lumen. Our *Case* 4 reported here is almost a replica of this case. The calcification in the wall was dense and the wall was rock-like and almost unindentable. The calcification can easily be seen in the X ray (*Fig.* 158). *Case* 5 was also similar.

That the pulmonary artery or its major branches are not immune from this pathologic process has been mentioned in the literature (Ask-Upmark, 1954; Barker and Edwards, 1955). In our *Case* 7 it will be seen that in addition to non-visualization of the left common carotid and the left subclavian artery and narrowing of the innominate artery, the upper branch of the right pulmonary artery was not visualized at all in any of the angiography radiographs, although the other branches of the right and left pulmonary arteries were very clearly delineated (*Fig.* 164). This clearly lends support to the contention that the pathologic process, whatever the aetiology, may involve different segments of vessels in the same case or in different cases and it will be wrong to expect the clinical pattern of the disease to conform to a particular type. Conceivably, the pulmonary arterial affection, if unchecked, may produce symptomatology of a very interesting type. The same pathologic process evidently can involve the abdominal aorta or branches thereof. Derrick, Pollard, and Moore (1959) recently described the arteriosclerotic pattern in such cases. Shaw and Maynard (1958) described cases with malabsorption syndrome, who were found at operation to have similar vascular insufficiency affecting the cœliac and superior mesenteric arteries. Harbitz (1926) reported a case of abdominal aorta where there was diffuse infiltrative lesion with cartilaginous consistency. Involvement of the renal artery was also reported by Ask-Upmark (1954). Our *Case* 3 had marked narrowing of the upper abdominal aorta with significant pressure gradient. There was also considerable thickening around the orifices of the large abdominal branches (*Fig.* 157). Following catheter aortography, she complained of acute abdominal pain and died suddenly in the ward. It may be that the trauma of the aortography procedure tipped the scales in already ischæmic areas affecting the alimentary tract, and precipitated her death.

Can we offer any rational explanation of the pathologic process involved from the admittedly limited material in this series? There are a few striking features amongst the cases reported here. Of the 10 cases, as many as 7 were young women and all cases were in the age-groups between 10 and 35 years, the large majority being between 15 and 25 years. Both these findings have been corroborated in other publications. In a number of cases there was history of febrile episodes prior to the development of the full-fledged disease complex and in a few there was history of joint pains. Almost all cases showed evidence of anæmia which in some responded to the corticosteroid group of drugs. Histology of the arteries in our series of cases was available in 7 cases. In 4 cases, biopsies were taken from affected vessels at the time of operation. In 1 case, biopsy was taken from an intercostal artery and in 2 cases studies were made after autopsy.

The histology of the affected arteries showed predominantly features of arteritis affecting the medial coat associated with intimal thickening. The nature and intensity of the arteritis differed in individual cases.

Inflammatory cellular exudate, often focal in distribution associated with focal disruption, was the most characteristic feature. In most cases there were changes in the ground substance with excess of acid mucopolysaccharide. Presence of hæmato-oxophylic substance in the media which histochemically was akin to similar substance associated with rheumatic arteritis was the outstanding feature in 2 cases. Giant-cell arteritis was a very characteristic feature in 2 cases. The atheromatous changes appeared to be secondary to the medial lesion.

The changes in the artery are suggestive of primary affection of the connective tissue resulting in arteritis. The presence of hæmato-oxophylic substance in 2 cases and giant-cell arteritis in 2 cases is strongly in favour of such a view. It is well recognized that tissue sensitization associated with connective-tissue disorder can be brought about by a rich variety of primary aetiological factors.

All these changes may appear to be of a non-specific nature, but it is evident that there is at least a strong bias in favour of rheumatic aetiology. The sex and the age of the patients, the history of febrile episodes, the history of joint pains in some cases are very suggestive features.

The surgical treatment of occlusive diseases of the aorta and its branches has made great progress in recent years. If the patient presents for treatment before irreversible complications have already set in, much can be done to alleviate the condition. For cases affecting the roots of the head vessels either thrombo-endarterectomy or end-to-side by-pass graft procedures can be employed. Endarterectomy is used when the disease is localized and more extensive lesions are treated by by-pass procedures (De Bakey and others, 1959; Bahnson and others, 1959; Davis and others, 1956). We have had no experience yet of operating on occlusion of the distal portions of the carotid or of vertebral arteries, but Rob and Wheeler (1957) and Eastcott, Pickering, and Rob (1954) have reported on their experiences in this type of disease in England, and De Bakey and others (1959) presented his results and spoke about his great experience in this type of occlusive disease in a recent meeting of the Association of Surgeons of India. Amongst other ingenious methods used in De Bakey's series were replacement of parts of diseased segments of vessels by patch dacron graft, total replacement of parts of diseased vessels by dacron tubes, etc. Accurate localization of the site and extent of the disease process is imperative to

obtain the best results from surgical procedures and here the value of good angiographic visualization cannot be overestimated. Our angiographic studies have been mostly of the venous type, the radio-opaque dye being introduced in the vein very rapidly as a bolus, and with good timing and proper changing of cassettes good visualization of the aorta and all its branches can be obtained. The procedure is easy and saves much time for the doctor and trouble for the patient.

For occlusive processes affecting the descending thoracic aorta, the by-pass procedure seems preferable to thrombo-endarterectomy. Three of our cases were treated in this way. Thrombo-endarterectomy has also been done and reported in the literature but is likely to weaken the wall and lead to insecurity.

The prognosis of the disease is uncertain. It is a progressive lesion, but the rate of progress varies in different cases. It is apparent that the disease may take effect and be well established at an early age. Our youngest patient with well-marked aortic occlusion was 10 years old. In some cases, significant occlusive signs and symptoms become well established during the course of 6–8 months (*Case* 8). If allowed to progress, blindness, hemiplegia, or other dramatic complications supervene. In some cases the patient develops left ventricular failure. It is clear, therefore, that the obstructive lesion must be tackled early before complications set in. As the obstruction is mechanical, it should be corrected by mechanical means, and fortunately advances in vascular surgery have made such corrective operations possible.

SUMMARY

A review is made of acquired occlusive diseases of the aorta and its branches. Ten cases studied between 1953 and 1960 are presented. Four of these cases were seen in the last 6 months. It is suggested that the disease is not such a rare entity as is generally believed.

It is emphasized that the disease may have variegated patterns, that affection of the head vessels may be associated with marked narrowing and constriction of the thoracic aorta, and that the abdominal aorta or its branches may also be affected, producing thereby symptoms referable to the alimentary tract. Evidence is presented to indicate that the pulmonary artery or its branches may also be affected along with affections of the aorta or the head vessels.

The possibility of rheumatic affection as the aetiological agent is strongly suggested. This belief is based partly on the clinical features of the cases (the age incidence, the sex, the febrile episodes, occasional joint pains, and anæmia) and partly on the histological features.

It is emphasized that the disease is a progressive lesion and that the progress may be rapidly downhill. Once the obstructive factor is established, efforts must be made to remedy it by operation. Fortunately, in most cases this can be accomplished and the patients can be greatly palliated.

Acknowledgements.—I am grateful to Dr. Michael De Bakey for generously sending us a supply of his dacron grafts. His visit to this country was very stimulating. Professor B. K. Aikat was responsible for nearly all the histologic studies. I am grateful to him for taking so much interest in this study. Dr. D. Sengupta skilfully performed all the pre-operative angiographic studies. Mr. S. Dutta, artist, is responsible for the illustrations. Thanks are also due to the Superintendent, S.S.K.M. Hospital, for the use of the hospital records.

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'SPONTANEOUS' RUPTURE OF THE DISTAL LARGE BOWEL

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SPONTANEOUS rupture of the rectum and pelvic colon is not a common condition, but when it occurs it requires immediate recognition and treatment. Six cases are reported and are compared with 19 similar cases in the literature.

HISTORICAL NOTE

Benjamin Brodie in 1827 described the case of a middle-aged woman whose rectum suddenly ruptured, apparently spontaneously. Huntley, in 1919, recorded the case of an army officer, aged 34, whose pelvic colon ruptured during defæcation. Both of these patients died, and in neither case was there any abnormality, apart from the rupture found in the bowel at post-mortem. Burckhardt (1908) appears to have been the first to deal successfully with this condition. His patient, a youth aged 17, developed acute abdominal symptoms during defæcation. At operation, a hole in the anterior wall of the rectum was sutured.

KNOWN CAUSES OF PERFORATION IN THE LARGE BOWEL

Perforation of the colon or rectum is commonly due to pathological states such as diverticulitis, tumour, or, more rarely, simple ulcer. Less frequently, it is the result of trauma such as may be caused by enemata, foreign bodies, faulty instrumentation, or external violence. Severe muscular strain, as when lifting a heavy load, sometimes results in a rupture of the small intestine and it is interesting to find that there is usually an associated inguinal hernia (Aird, 1935; Wilensky and Kaufman, 1937). No similar case of rupture of the large bowel has been reported. A sudden rupture of the rectum may complicate a rectal prolapse (Lindenbaum, 1936), and this may be followed by a protrusion of a loop of small intestine through the tear (McLanahan and Johnson, 1945). In some cases, however, as in those described by Brodie and by Huntley, no apparent cause can be found and it is this group which forms the subject of the present communication. The following 6 cases illustrate the nature of the problem.

CASE REPORTS

Case 1.—Mrs. G. H., aged 52 years, was admitted to hospital as an emergency, complaining of severe abdominal pain. The pain had begun quite suddenly at 8 p.m. on the previous day while she was attempting, unsuccessfully, to move her bowels. She retired to bed and the pain continued throughout the night. The next day she called in her doctor who sent her to hospital with the diagnosis of acute appendicitis. Prior to this attack the patient had been in good health; there was no history of constipation.

ON EXAMINATION.—The patient, an obese woman, was flushed and her tongue coated. Her pulse-rate was 70, temperature 97·8, and blood-pressure 135/75. There was generalized abdominal tenderness and guarding, which was most marked in the right iliac fossa. A diagnosis of acute appendicitis was made and operation advised.

AT OPERATION.—The peritoneal cavity contained a small amount of purulent fluid, but the appendix was normal. A localized inflammatory area was found in the pelvic colon in the centre of which was a small perforation. The remainder of the large intestine was normal and no foreign body was found. The perforation was closed with interrupted linen thread sutures, the peritoneal cavity drained, and the wound closed.

POST-OPERATIVE PROGRESS.—The patient made a straightforward recovery and was discharged home three weeks after admission. During convalescence a sigmoidoscopy and barium enema were done, which did not show any abnormality in the colon or rectum. Six months later the patient had some rectal bleeding. She was readmitted to hospital and a further sigmoidoscopy and barium enema were done but no abnormality was found. Two years later she was well and had no complaints.

Case 2.—D. S., a youth aged 16 years, previously healthy, was admitted as an emergency to hospital, complaining of severe generalized abdominal pain which had awakened him in the early morning. It persisted throughout the forenoon and later he complained of pain in both shoulders. He vomited several times. There was no history of constipation.

ON EXAMINATION.—The patient was pale and ill, pulse-rate 100, temperature 99·8. There was generalized abdominal tenderness and rigidity. Digital examination of the rectum revealed tenderness anteriorly, there was no mass and the rectum was empty. A diagnosis of peritonitis due to a perforated viscus was made and an immediate laparotomy done.

AT OPERATION.—Gas and purulent fluid escaped as the peritoneal cavity was opened. The appendix was normal and there was no Meckel's diverticulum. On the anterior wall of the rectum, just above the peritoneal reflection, there was an oval hole measuring about ¼ in. in length. The edges of the perforation were a little ragged, there was no bleeding, and there were only minimal signs of inflammation. The remainder of the large intestine was normal. The hole was closed transversely in two layers, using interrupted linen thread sutures. The fluid was removed from the peritoneal cavity and the wound closed around a drain to the posterior rectus sheath.

POST-OPERATIVE PROGRESS.—The patient made a straightforward recovery from the operation. A subsequent sigmoidoscopy did not show any abnormality in the lower colon or rectum. A barium enema showed a normal colon and rectum. He was questioned on the possibility of the injury being due to rectal interference, but denied any such possibility. Two years after operation he was well.

Case 3.—Mrs. W. W., aged 55 years, who weighed 15 stone and was suffering from increasing constipation, was admitted to hospital with the history that, whilst straining at stool seven days previously, she was seized with severe pain in the lower abdomen. Immediately following the onset of pain she felt ill, vomited, and retired to bed.

During the next few days the pain persisted and her general condition remained poor. She was therefore referred to hospital for observation.

On Examination.—Pulse-rate 76, temperature 98, blood-pressure 130/80. There was abdominal distension and some tenderness, particularly below the level of the umbilicus. Fæces could be felt in the pelvic colon.

The day after admission, the patient had an attack of diarrhœa; following this, she had some relief from the abdominal pain. A laparotomy was done one week after admission.

At Operation.—The abdomen was explored through a lower right paramedian incision. The large intestine, which contained firm lumps of fæces, was distended and hypertrophied; no diverticula were seen. A small abscess was found in the pelvis and on emptying this, a transverse tear ½ in. long was seen in the anterior wall of the rectum, just above the peritoneal reflection. The edges of the tear were thin and necrotic. There was no other abnormality in the rectum, which was empty. The hole was closed in two layers, the pelvis drained, and a transverse colostomy done.

Post-operative Progress.—The patient made a good recovery from the operation and four months later the colostomy was closed. A sigmoidoscopy which was done during convalescence showed no abnormality in the rectum or lower colon.

Case 4.—Mr. O. W. P., aged 59 years, was admitted to hospital complaining of abdominal pain which had begun suddenly after he had moved his bowels earlier in the day. Immediately after the onset of the pain, he experienced a bearing-down sensation and passed a few drops of blood per rectum. The abdominal pain, which had at first been in the lower abdomen, soon became generalized, and was also felt in the right shoulder. He felt sick and vomited once or twice.

On Examination.—The patient was in a state of shock; he was cold, sweating, and his lips were cyanosed. The abdomen was diffusely tender and rigid. There was an easily reducible right inguinal hernia. A hard mass of fæces was felt in the rectum on digital examination. After resuscitation, a laparotomy was done, the presumptive diagnosis being peritonitis due to a perforated duodenal ulcer, since he gave a history of peptic ulceration.

At Operation.—The abdomen was opened through an upper right paramedian incision. The peritoneal cavity contained some cloudy yellow fluid. There was scarring of the duodenum due to previous ulceration, but there was no perforation. The upper paramedian wound was extended downwards and the pelvis examined. Lumps of fæces were found lying free on the pelvic floor. A ¾-in. long tear was found in the antimesenteric border of the pelvic colon at the pelvi-rectal junction. The hole was closed in two layers using interrupted sutures of catgut. A transverse colostomy was made, the pelvis drained, and the wound closed.

Following operation the patient developed a lung infection and his abdominal wound broke down on two occasions. His general condition remained very poor and he died in circulatory failure, four weeks after operation.

Post-mortem Examination.—Apart from a small benign polyp in the descending colon, there was no macroscopic evidence of any disease at the site of rupture in the pelvi-rectal area, or elsewhere in the colon or rectum.

Case 5.—Mrs. L. L., aged 65 years, was admitted to hospital as an emergency, complaining of severe abdominal pain. About four hours before admission she was washing the dishes, following a good lunch, when she felt a desire to move her bowels. Whilst in the act of defæcation, but not straining, she was seized with a sudden and severe pain over the whole abdomen; soon afterwards she vomited. She retired to bed, called her doctor, and was sent to hospital. Apart from a history of chronic constipation, the patient had been in good health.

On Examination.—The patient was cold, shocked, and in severe pain; pulse-rate 84, temperature 98·6, blood-pressure 75/50. Her tongue was dry and a little coated. There was generalized abdominal tenderness and rigidity which was most pronounced in the lower abdomen. No bowel sounds were heard. The patient was considered to be suffering from peritonitis due to a perforated viscus and a laparotomy was done.

At Operation.—The abdomen was opened through a right para-umbilical incision. Brown fæcal-smelling fluid was present in the peritoneal cavity and several round lumps of fæces were found lying free in the pelvis. On the anterior wall of the pelvic colon, adjacent to the pelvi-rectal junction, there was a 1½-in. long tear through all coats of the bowel wall. The tear was recent and there was little evidence of inflammation at the edges. The remainder of the colon was normal and was loaded with round hard lumps of fæces. No cause for the rupture was found. The hole was closed in two layers using catgut sutures. The pelvis was drained and a left iliac colostomy done. She made a slow but complete recovery.

A follow-up sigmoidoscopy and barium enema did not show any abnormality in the colon or rectum. When last seen, the patient was well and awaiting closure of her colostomy.

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During the next few days the pain persisted and her general condition remained poor. She was therefore referred to hospital for observation.

ON EXAMINATION.—Pulse-rate 76, temperature 98, blood-pressure 130/80. There was abdominal distension and some tenderness, particularly below the level of the umbilicus. Fæces could be felt in the pelvic colon.

The day after admission, the patient had an attack of diarrhœa; following this, she had some relief from the abdominal pain. A laparotomy was done one week after admission.

AT OPERATION.—The abdomen was explored through a lower right paramedian incision. The large intestine, which contained firm lumps of fæces, was distended and hypertrophied; no diverticula were seen. A small abscess was found in the pelvis and on emptying this, a transverse tear ½ in. long was seen in the anterior wall of the rectum, just above the peritoneal reflection. The edges of the tear were thin and necrotic. There was no other abnormality in the rectum, which was empty. The hole was closed in two layers, the pelvis drained, and a transverse colostomy done.

POST-OPERATIVE PROGRESS.—The patient made a good recovery from the operation and four months later the colostomy was closed. A sigmoidoscopy which was done during convalescence showed no abnormality in the rectum or lower colon.

Case 4.—Mr. O. W. P., aged 59 years, was admitted to hospital complaining of abdominal pain which had begun suddenly after he had moved his bowels earlier in the day. Immediately after the onset of the pain, he experienced a bearing-down sensation and passed a few drops of blood per rectum. The abdominal pain, which had at first been in the lower abdomen, soon became generalized, and was also felt in the right shoulder. He felt sick and vomited once or twice.

ON EXAMINATION.—The patient was in a state of shock; he was cold, sweating, and his lips were cyanosed. The abdomen was diffusely tender and rigid. There was an easily reducible right inguinal hernia. A hard mass of fæces was felt in the rectum on digital examination. After resuscitation, a laparotomy was done, the presumptive diagnosis being peritonitis due to a perforated duodenal ulcer, since he gave a history of peptic ulceration.

AT OPERATION.—The abdomen was opened through an upper right paramedian incision. The peritoneal cavity contained some cloudy yellow fluid. There was scarring of the duodenum due to previous ulceration, but there was no perforation. The upper paramedian wound was extended downwards and the pelvis examined. Lumps of fæces were found lying free on the pelvic floor. A ¾-in. long tear was found in the antimesenteric border of the pelvic colon at the pelvi-rectal junction. The hole was closed in two layers using interrupted sutures of catgut. A transverse colostomy was made, the pelvis drained, and the wound closed.

Following operation the patient developed a lung infection and his abdominal wound broke down on two occasions. His general condition remained very poor and he died in circulatory failure, four weeks after operation.

POST-MORTEM EXAMINATION.—Apart from a small benign polyp in the descending colon, there was no macroscopic evidence of any disease at the site of rupture in the pelvi-rectal area, or elsewhere in the colon or rectum.

Case 5.—Mrs. L. L., aged 65 years, was admitted to hospital as an emergency, complaining of severe abdominal pain. About four hours before admission she was washing the dishes, following a good lunch, when she felt a desire to move her bowels. Whilst in the act of defæcation, but not straining, she was seized with a sudden and severe pain over the whole abdomen; soon afterwards she vomited. She retired to bed, called her doctor, and was sent to hospital. Apart from a history of chronic constipation, the patient had been in good health.

ON EXAMINATION.—The patient was cold, shocked, and in severe pain; pulse-rate 84, temperature 98·6, blood-pressure 75/50. Her tongue was dry and a little coated. There was generalized abdominal tenderness and rigidity which was most pronounced in the lower abdomen. No bowel sounds were heard. The patient was considered to be suffering from peritonitis due to a perforated viscus and a laparotomy was done.

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Table II.—Summary of Previously Reported Cases of 'Spontaneous' Rupture of Distal Large Bowel

Case No.	Author	Sex	Age	Possible Aetiological Factors			Operation	Result	Remarks
				Constipation	Strain of Defæcation	Site of Rupture			
1	Brodie (1827)	F.	Middle age	None reported	No	Anterior wall of rectum	Laparotomy and reduction of small bowel which had prolapsed through the tear	Death	Post-mortem: No evidence of ulceration found in the neighbourhood of the tear. It was concluded that the opening was the result of accidental laceration
2	Burckhardt (1908)	M.	17	None reported	Yes	Anterior wall of rectum	Laparotomy and suture of hole	Recovery	
3	Hellström (1914)	M.	42	None reported	No	Anterior wall of rectum	Laparotomy and suture of hole	Death	Post-mortem: Septic peritonitis Microscopy: Infiltration of edges of rupture
4	Huntley (1919)	M.	34	History of chronic constipation	Yes	Pelvi-rectal junction	Laparotomy and suture. Peritoneal drain	Death	Post-mortem: No abnormal pathology found in the rectum
5	Kyaergaard (1920)	F.	53	History of constipation	No	Anterior wall of rectum	Laparotomy; ? suture	Death	Post-mortem: No anatomical changes in rectal wall
6	Küttner (1926)	M.	20	History of chronic constipation	Yes	Anterior wall of rectum	Laparotomy and suture	Death	Post-mortem: No anatomical changes in rectal wall
7	Kurkiala (1928)	M.	40	None reported	Yes	Anterior wall of rectum	Laparotomy and suture	Death	
8	Brown (1944)	M.	30	None reported	No	Antimesenteric border of pelvic colon, close to pelvi-rectal junction	Laparotomy and suture. Peritoneal drain	Recovery	At operation: Edges of tear irregular. Bowel wall macroscopically normal Follow-up: Barium enema normal
9	Allen (1948)	M.	54	History of chronic constipation	No	Anterior wall of rectum	Laparotomy and suture. Peritoneal drain. Left inguinal colostomy	Recovery	At operation: Slit in mucosa was not indurated or friable. No hæmorrhage. Rectum and colon otherwise normal Follow-up: Colostomy closed 3 months later
10	Lataix (1949)	F.	43	None reported	No	Anterior aspect of pelvi-rectal junction	Laparotomy and suture. Peritoneal drain. Left iliac colostomy	Recovery	Large lower abdominal incisional hernia present At operation: Bowel wall appeared macroscopically normal. No induration at edges of rupture Follow-up: Sigmoidoscopy normal. Colostomy closed after 3 months
11	Weinstein and Roberts (1952)	M.	63	At operation: Colon distended and packed with hard fæces	Yes	Antimesenteric border. Sigmoid flexure	Laparotomy and suture. Peritoneal drain. Cæcostomy	Recovery	At operation: Edges of tear were pouting and everted. Surrounding bowel wall normal Follow-up: Sigmoidoscopy and barium enema normal
12	Brearley (1954)	M.	66	History of chronic constipation At operation: Colon contained hard fæces	No	Antimesenteric border of pelvic colon	Laparotomy and suture. Exteriorization of affected loop	Death	At operation: Edges of rupture necrotic
13	Gauble (1954)	M.	58	None reported	Yes	Pelvic colon	Laparotomy. Peritoneal and perineal drain. Proximal colostomy	Recovery	Patient had operation (conservative) for carcinoma of rectum 3 years previously At operation: Perforation felt, but not seen, behind the bladder
14	Eadie (1955)	F.	57	History of constipation At operation: Colon packed with hard fæces	Yes	Antimesenteric border of apex of pelvic colon	Laparotomy. Peritoneal drain. Exteriorization of affected loop	Recovery	At operation: Edge of rupture clean cut and healthy with normal mucosa pouting through. No inflammation. Remainder of bowel normal Follow-up: Colostomy closed 6 weeks later. Barium enema normal
15	Henderson (1957)	F.	38	On examination: Some firm scybalous fæces in the rectum At operation: Firm fæces in colon	No	Anterior surface of sigmoid colon	Laparotomy and peritoneal drain. Exteriorization of affected loop	Recovery	At operation: Very little inflammatory reaction at margin of defect Follow-up: Resection of affected loop—histology showed no significant changes. Patient well 5 years later

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Case No.	Author	Sex	Age	Possible Aetiological Factors			Operation	Result	Remarks
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16	Henderson (1957)	F.	49	History of chronic constipation At operation: Very hard fæcal matter in left colon	No	Antimesenteric surface of left colon near rectosigmoid junction running down into rectum	Laparotomy and suture. Proximal colostomy	Recovery	At operation: No evidence of inflammation of more than 24 hours' duration Follow-up: Colostomy closed. Barium enema normal
17	Rapp (1958)	F.	69	History of constipation On examination: Rectum full of fæces	No	Right side of sigmoid colon	Laparotomy and suture. Transverse colostomy	Death	Post-mortem: Colon contained many fæcal boluses. No gross or microscopical evidence of disease in colon
18	Rapp (1958)	F.	65	At operation: Hard fæcal masses in pouch of Douglas	No	Antimesenteric border of sigmoid colon 3 in. above peritoneal reflection	Laparotomy and suture. Transverse colostomy	Recovery	At operation: No macroscopic changes seen in colon Follow-up: Barium enema showed some narrowing of sigmoid colon, otherwise no abnormality
19	Cronin (1959)	F.	69	At operation: Colon filled with hard fæces	No	Antimesenteric border of apex of sigmoid colon	Laparotomy and exteriorization of affected loop	Death	Post-mortem: Histological sections through the site of rupture showed no abnormality in the bowel

anterior longitudinal bundle whose edges had retracted to leave an area of linear weakness which ultimately gave way. This recalls to mind the longitudinal tears which develop in the anterior longitudinal bundle of the over-distended cæcum in cases of acute obstruction of the large bowel. In the authors' two rectal cases the gap was oval in one and a transverse slit in the other. Each involved the anterior wall where the longitudinal coat had broadened out and the amount of retraction was correspondingly less. The operation records are, however, insufficiently complete in most of the rectal cases to confirm that this type of lesion is characteristic of ruptures in the rectum.

3. Clinical Features.—

Age and Sex do not appear to have any significance since the patients' ages ranged from 16 to 69 years, and since 13 were females and 12 were males.

Constipation.—In 16 cases the patient either complained of constipation or some evidence of it was found on clinical examination or at operation. In the other cases, where no mention was made of constipation, its presence may have been overlooked.

Defæcation.—In 11 cases there was a history of defæcation at or immediately preceding the onset of pain. It is an interesting fact that defæcation is the only type of strain which has resulted in a spontaneous rupture of the large bowel. It is also noteworthy that the lesion occurs within that part of the large bowel which is active during defæcation. Severe muscular effort has ruptured the rectum only when it is already prolapsed. It therefore appears that the strain of defæcation has its own peculiarities which under certain conditions may result in spontaneous rupture.

THE MECHANISM OF RUPTURE

A consideration of these aetiological factors suggests that they combine to produce a lesion in the distal colon or upper rectum which is mechanical in origin.

1. Thus it appears that the rupture may occur at the apex of the sigmoid loop, if when loaded with fæces it becomes over-distended by peristaltic waves which are unable to force its solid and gaseous contents through the pelvi-rectal junction. This accident will be more likely to happen if the patient is constipated and in the act of defæcation.

2. Again the pelvi-rectal junction is a part of the colon which can become suddenly and completely obstructed by kinking, abnormal fixation, prolonged spasm, or fæcal impaction. In the presence of one of these mechanical factors a hard scybalous mass could produce a ball-valve effect, or an impaction which might lead, under vigorous peristalsis, to a localized split in the gut at that site.

3. Similarly, should a hard fæcal bolus become arrested in the upper part of the rectum or at one of its valves during defæcation, the thrust of peristalsis might lead to a rupture in its anterior wall.

The voluntary contraction of the abdominal muscles which normally helps to initiate defæcation may be accentuated in the presence of constipation and thus increase still further intracolic pressure and the risk of spontaneous rupture.

DIAGNOSIS

The rupture is usually marked by the onset of acute lower abdominal pain which spreads quickly to the rest of the abdomen and may be felt in the shoulders. It may come on dramatically during defæcation or there may be no history of strain. It has even occurred during sleep. In only one case has rectal bleeding been recorded.

On examination the patient is pale, anxious, and in obvious pain, and is usually shocked. The abdomen is tender and rigid, particularly below the umbilicus, and there may be free gas in the abdomen. Rectal tenderness is probable and hard fæces may be felt in the iliac colon or rectum. A straight X-ray examination may reveal the presence of free gas. The diagnosis is likely to be that of a perforated appendix, peptic ulcer, or colonic diverticulum, or of peritonitis following a perforation, but laparotomy will reveal the exact diagnosis.

TREATMENT

After resuscitation a laparotomy should be undertaken. Turbid or purulent fluid containing flakes of fæces may be present, or more solid fæces may lie in the pelvis or be seen escaping from the distal colon or rectum. Examination of the stomach, duodenum, and appendix will show no causal abnormality. There may be scybalous masses in the colon or rectum and eventually the rupture will be found and its nature determined. The large bowel wall will appear healthy except where delay has been followed by inflammation or a pelvic abscess has formed. Later still, there may be a generalized peritonitis.

Treatment consists in removing the infected fluid and fæces and in closing the tear in the bowel, usually by two layers of sutures. If the bowel contains solid fæces these should be moved away from the site of rupture to permit suture without tension. It is always safer in such a case and when the proximal colon is distended, to perform a temporary colostomy or a cæcostomy. If the colon or rectum has emptied, a proximal colostomy need not be done, but it is a wise precaution to pass a rectal tube to keep the site of repair free from tension. When the site of rupture is at the apex of the sigmoid loop, an alternative procedure is to exteriorize this loop as a colostomy. This offers a rapid solution of the immediate problem, particularly when the proximal colon is loaded or distended. Peritoneal drainage for a few days is desirable whenever operation has been delayed for twelve hours or more after the rupture. The wound, if contaminated, may also require drainage. Following operation, gastric suction and intravenous therapy for one or two days will minimize the risk of paralytic ileus and an antibiotic should be given to counteract peritoneal infection.

It has been our practice to re-examine the patients by sigmoidoscopy and barium enema some weeks after convalescence to ensure that no hidden pathology has been overlooked. So far, these examinations have all confirmed the accidental nature of these spontaneous ruptures.

MORTALITY

Ten of the 25 cases reviewed in this paper died after operation. Of the 7 reported in the literature up to 1928, only 1 survived. Of the 18 which have occurred since 1944, only 4 have died. This marked improvement is no doubt due to modern methods of treatment, but the mortality is still too high and this emphasizes the seriousness of the condition and the need for haste and alertness in dealing with it.

SUMMARY

Six new cases of spontaneous rupture of the normal distal large bowel are described and 19 cases from the literature are reviewed, bringing the total of recorded cases to 25.

The aetiology of these cases is discussed. The ruptures occur in the region of the pelvi-rectal junction, in the sigmoid loop, and in the upper rectum. The tear is considered to be mechanical in origin and to be due to a sudden rise in intraluminar pressure which results from sudden obstruction during active peristalsis. Rupture may occur quite spontaneously but is more likely to occur in the presence of constipation and during defæcation. The diagnosis, treatment, and mortality are also discussed.

We are indebted to Mr. Robert V. Cooke for sending us the details of *Case* 6 and to Mr. D. C. Dickson for allowing us to publish the records of *Case* 5.

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PRIMARY TUMOURS OF THE URETER

A REPORT OF 9 CASES

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PRIMARY tumours of the ureter are rare. Riches was able to collect only 21 cases from centres in the British Isles for the Congress of the British Association of Urological Surgeons in 1951. Whitlock, McDonald, and Cook (1955) reported 33 cases treated at the Mayo Clinic up to 1945. Mortensen and Murphy (1950) collected 245 cases of which 21 were benign, and Abeshouse (1956) found 454 malignant cases and 138 benign cases in the world literature. The case histories of 9 patients seen since 1950 are submitted.

FIG. 170.—*Case* 1. Ureterogram with filling defect below pelvic brim.

CASE REPORTS

Case 1.—Female, aged 70 years, seen in June, 1950, on account of hæmaturia and left lower quadrant pain. Fifteen months previously she had had a left nephrectomy for hydronephrosis at another hospital. The hæmaturia had been present for some months before the operation, and blood was seen emerging from the left ureteric orifice. An intravenous pyelogram (I.V.P.) had revealed a renal-type hydronephrosis. No mention was made in the notes of any dilatation of the ureter either on pyelography or at operation. No retrograde examination was performed. On passing a catheter up the left ureteric stump old and fresh blood emerged. A ureterogram (*Fig.* 170) revealed a filling defect just below the pelvic brim. The tumour was palpable bimanually and mobile.

Operation: Ureterectomy.

The specimen was a pedunculated papilloma about 3 cm. in length. No seedlings were present. Histology: Transitional-celled benign papilloma.

Case 2.—C. F., male, aged 74 years. First seen in May, 1955, with painless hæmaturia for one week. The right kidney did not show up well in any of the I.V.P. pictures. The left kidney was normal. No bleeding followed the introduction of a ureteric catheter, and retrograde pyelography revealed what appeared to be a small filling defect in the region of the middle calyx. The ureter was very irregular but no filling defect was observed (*Fig.* 171).

A right nephrectomy was performed under the impression that the bleeding was due to a hæmangioma. No dilatation of the ureter was found at operation. Section of the kidney revealed no tumour. The patient was next seen 3 years later. He had been complaining of pain in the right lower quadrant for some time and had a week's hæmaturia. A hard, palpable mass was present at the right pelvic brim, and on exploration this was found to be an inoperable tumour of the ureter.

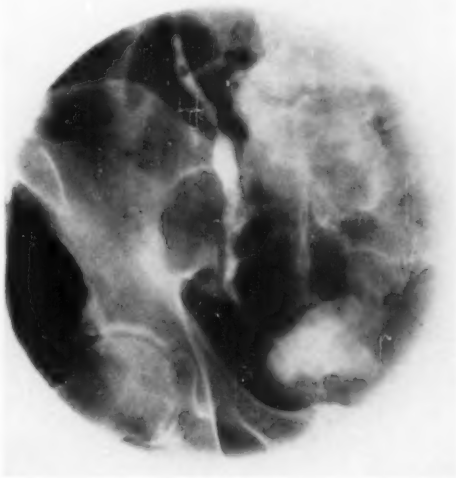

FIG. 171.—*Case* 2. A retrograde pyelogram showing marked irregularity of the ureter which was attributed to over-filling of the pelvis. Tumour found three years later at pelvic brim.

Biopsy: Transitional-celled growth. The patient died four months later, and post-mortem revealed secondaries in the lungs and liver.

Comment: The irregularity of the ureter was attributed erroneously to over-filling of the renal pelvis and the local dilatation in the middle third was overlooked.

Case 3.—Male, aged 56 years. First seen in July, 1957, with three days' left loin pain and hæmaturia. I.V.P.: No dye on the left side. Cystoscopy: Nil. Ureteric catheter could not be passed into the left renal pelvis. No bleeding followed its introduction. Pyelography: Left hydronephrosis with incomplete filling of the pelvis (*Fig.* 172). The upper ureter in its first 6 cm. was not outlined. Diagnosis: Ureteric tumour.

Operation: Nephro-ureterectomy. The tumour was about 3 cm. below the uretero-pelvic junction and was about the size of an acorn (*Fig.* 173). It was hard and firm,

but there was no fixation to the peri-ureteric tissues. The tumour was about 1·5 cm. long and the lumen of the ureter would just admit a fine probe.

Histology: Transitional-celled carcinoma poorly differentiated.

FIG. 172.—*Case* 3. The upper ureter is very faintly outlined at the tumour site.

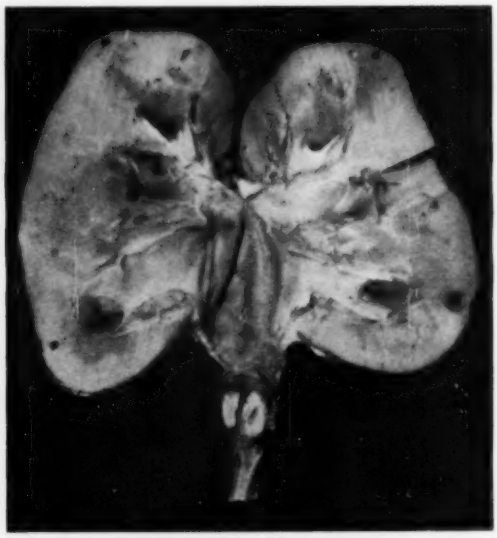

FIG. 173.—*Case* 3. Sessile invasive ureteric tumour just below the uretero-pelvic junction.

High-voltage therapy was instituted, but the patient died in May, 1958, of multiple secondaries.

Case 4.—Male, aged 69 years. Attended hospital in March, 1955, complaining of 3 weeks' painless hæmaturia. I.V.P.: Right, kidney normal; left, kidney hydronephrotic

FIG. 174.—*Case* 4. The lower end of the ureter is full of growth with a seedling at the orifice. Kidney grossly hydronephrotic.

with dilatation of the ureter down to the pelvic brim where a filling defect was noticed.

Diagnosis: Ureteric tumour. Operation declined. Patient seen again in November, 1958, on account of acute retention. I.V.P.: No dye on the left side. Cystoscopy: Enlarged prostate. Bladder tumour about the size of a golf ball in the neighbourhood of the right ureteric orifice. Ureteric catheter blocked on the left side just inside the ureteric orifice.

FIG. 175.—*Case* 5. Cup-shaped filling defect with complete obstruction of the ureter.

Operation: Partial cystectomy and left nephroureterectomy with cuff of bladder. The cystotomy opening was extended as a racket incision to embrace the intramural ureter.

Histology: The lower part of the ureter contained a firm, greyish tumour. It was fixed to the inner aspect of the wall in several places. The intramural ureter contained a separate tumour (*Fig.* 174). Both tumours were transitional-cell growths. The bladder tumour (T.1) was a solid and papillary well-differentiated carcinoma. The kidney parenchyma was almost completely atrophied.

This patient was kindly referred by Mr. A. J. Walton who originally made the diagnosis.

Case 5.—Female, aged 77 years. Attended hospital in November, 1959, with right renal pain which had been present for 1½ years. Right kidney large and tender. I.V.P.: No dye right side. Ureteric catheter blocked 5 cm. below uretero-pelvic junction. No bleeding followed its introduction. Ureterogram: No dye reached the kidney. Tumour of ureter diagnosed (*Fig.* 175).

Operation: Perinephric abscess. Tumour of ureter found 6 cm. below uretero-pelvic junction. Nephro-ureterectomy with cuff bladder.

Histology: Transitional-cell carcinoma.

Case 6.—Male, aged 48. Attended hospital in January, 1960, with a history of two weeks' painless hæmaturia. Intravenous pyelography: Nil. Cystoscopy: Negative.

FIG. 176.—*Case* 6. I.V.P. Oval filling defect in the lower third of the left ureter. No hydro-ureter. No hydronephrosis.

Right retrograde pyelogram: Negative. A catheter could not be inserted for technical reasons into the left ureteric orifice. Repeat I.V.P.: Right kidney normal. Filling defect at the lower end of the left ureter (*Fig.* 176). Left uretero-pyelogram: Confirmed the filling defect. Bi-manual examination: Tumour not felt.

Operation: Nephro-ureterectomy with bladder cuff. Tumour freely mobile. Section of tumour: Transitional-celled non-invasive carcinoma, 2 × 1 cm. No gross abnormality of kidney.

Case 7.—N. L., female, aged 41 years. First seen in July, 1957, with a T.1 tumour of the bladder. This was a transitional-celled carcinoma. The tumour was excised and radio-active gold grains implanted. An I.V.P. at that time revealed no abnormality in the upper urinary tract. Repeated check cystoscopies were carried out.

In November, 1958, a tumour was seen in the roof of the bladder at the site of the cystotomy. This was excised with the abdominal scar. It was also a T.1 transitional-celled tumour.

The follow up was regular, and in March, 1960, a papilliferous growth was found emerging from the left

FIG. 177.—*Case* 7. Gross hydronephrosis and hydro-ureter. Filling defect just outside the bladder. Gold grains in situ from the previously treated cancer of the bladder.

ureteric orifice. An I.V.P. showed a non-functioning left kidney and a ureteropyelogram a filling defect in the juxta vesical ureter (*Fig.* 177).

A nephro-ureterectomy was performed with a cuff bladder. Dissection of the lower ureter was difficult owing to the previous radiation.

Histology: Transitional-celled carcinoma confined to the mucosa.

Case 8.—Mrs. L., aged 65 years. First seen in January, 1960, with hæmaturia. There was pain in the left renal area and frequency of micturition. The left kidney was enlarged and tender.

I.V.P.: No dye on the left side. Cystoscopy: Nil. Ureteric catheter blocked 1 cm. inside the ureteric orifice. No bleeding. All dye returned into the bladder and a retrograde was impossible.

Aortogram: Left hydronephrosis.

Operation (April, 1960): Nephrectomy. The ureter was very dilated and was the size of an index finger. It was followed to the pelvic brim where a tumour was discovered. Left ureterectomy with bladder cuff.

Histology: Tumour attached to the ureteric wall over a distance of 5 cm. Lumen almost completely obstructed. Transitional-celled carcinoma. Kidney hydronephrotic with several surface cysts: chronic pyelonephritis (*Fig.* 178).

Case 9.—R. C. Male, aged 59 years, was seen in January, 1960, with painless hæmaturia which he had had for one week.

I.V.P.: Left kidney normal. Right kidney: Renal type hydronephrosis. Retrograde pyelogram: Catheter obstructed after a few centimetres. No bleeding. Filling defect just below the pelvic brim (*Fig.* 179). Operation: Nephro-ureterectomy with cuff of bladder. A pedunculated tumour (*Fig.* 180).

Histology: Transitional-celled carcinoma. No invasion of mucosa.

FIG. 178.—*Case* 8. Tumour completely filling ureter.

FIG. 180.—*Case* 9. Pedunculated tumour with bladder cuff.

DISCUSSION

According to Scott (1943) the greatest incidence of the disease is in the sixth and seventh decades. In this group the youngest was 41 and the oldest 77 years. The youngest case on record was of a benign tumour in a boy of 10 reported by MacLean and Fowler (1956).

Sixty-six per cent of the tumours were found in the lower third of the ureter (MacLean and Fowler, 1956). In this series 2 were in the upper, 1 in the middle, and 6 in the lower third. The tumour may

FIG. 179.—*Case* 9. Filling defect in the lower third of the ureter, with dilatation above it, and hydronephrosis.

be single or multiple, but seedlings are only associated with the papillary type (*Case* 4). Stasis as a factor in the production of urinary cancer has been stressed by Scott and Boyd (1953), and this may explain the predominance of the juxtavesical ureter as the commonest site of these neoplasms.

There are two main groups: papillary about 60 per cent and non-papillary 40 per cent of the total. The stalk of the papillary tumour may be broad or narrow and it may be several centimetres in length. In this group of 9 reported cases the tumour was a transitional-cell growth, and Crassweller (1958) states that well over 50 per cent were of this type. The other varieties are squamous adenocarcinoma, and mesenchymal. Eight cases of leiomyosarcoma have been noted, and 1 case of spindle-cell sarcoma was mentioned by Riches, Griffiths, and Thackray (1951). Four cases of hæmangioma were collected by Hamsher, Farrar, and Moore (1958), and 5 cases of bilateral primary carcinoma have been collected by Gracia (1958). The tumours may be single or multiple: and simple tumours can give rise to seedlings.

Mortensen and Murphy (1950) and Macalpine (1951) have drawn attention to the possibility of retrograde spread which is another point in favour of radical surgery.

Two of the 9 cases had bladder tumours. In *Case* 4 it was on the opposite side to that of the ureteric, and in *Case* 5 the bladder growth was on the same side but it was discovered 3 years earlier. Careful scrutiny of this patient's I.V.P.s showed no evidence of ureteric obstruction when the bladder growth was first observed. In the 33 cases mentioned by Whitlock and others (1955), 14 had bladder neoplasms, and all the growths of the ureter were of the papillary variety. In 10 other cases bladder tumours developed some years later. The necessity of check cystoscopy is therefore obvious in all cases of treated ureteric and bladder tumours.

The symptoms are hæmaturia and symptoms due to ureteric obstruction. The bleeding may be painless and occasional worm-like clots have been reported. When the ureter is grossly obstructed there is loin pain—*Cases* 2, 5, 7, and 8. Pain over the tumour (*Case* 3) is usually due to local extension, and there may be pain from secondaries.

DIAGNOSIS

Two cases were missed. In *Case* 4 the dilated ureter observed at nephrectomy was followed to the pelvic brim where the tumour was located. Diagnosis was made on intravenous pyelography in only two cases (in one of these it had to be repeated). A renal type of hydronephrosis was present in *Cases* 1 and 9, and in 5 no dye was seen in the kidney of the affected side. The classical sign of brisk hæmorrhage following the introduction of a ureteric catheter was present in only 2 cases (1 and 7). Too much reliance, therefore, should not be placed on intravenous pyelography or on the ease with which the ureteric catheter is passed without provoking hæmorrhage. The number of reports in the literature where the tumour was discovered some time after nephrectomy bears witness to this.

Whitlock and others (1955) report a case of a patient who had 9 cystoscopic diathermies before the ureteric tumour was found, and Stock and Wells (1948) record the history of a man whose hæmaturia persisted after prostatectomy and later after the removal of a bladder stone. A ureteric tumour, the cause of the bleeding, was finally discovered.

Uretero-pyelography is the surest diagnostic method. A Chevassu catheter should be used and the patient X-rayed on the operating table. A coloured opaque solution as suggested by Tresidder and Warren (1954) should be injected under vision so that regurgitation into the bladder can be observed. If regurgitation should occur the catheter can be adjusted or a larger one employed.

The nature of the filling defect varies with the type of tumour, and when it is sessile, only a trickle of dye may be visible in the narrowed lumen. In pedunculated growths the filling defect is cup-shaped and a moth-eaten appearance may be seen. Dilatation of the ureter above the growth is usual, but is not always present (*Case* 6). Dilatation of the ureter below the tumour is not uncommon and is due to propulsion of the growth from peristalsis. Confusion may arise if a stone is present above the tumour or if the tumour is covered with calcium salts. In these appearances the filling defect seen in the ureteropyelogram is larger than the opaque shadow seen on the scout films.

TREATMENT

The standard operation is a nephro-ureterectomy with a cuff of bladder. It was employed in 6 of the 9 cases as a one-stage procedure without operative mortality. Uretero-nephrectomy has been recommended by some writers so that the operability of the tumour can first be assessed. Colston (1935) suggests that in bad risk cases instead of removing the bladder cuff the mucous membrane of the intramural ureter should be destroyed by fulguration, and other writers suggest that a cystoscopic diathermy might be used for the same purpose. If the tumour is a squamous one Abeshouse thinks that removal of a bladder cuff is not necessary. Local excision has been done by Vest (1945) in 3 benign cases with satisfactory results. It is a dangerous procedure as diagnosis has to be established by frozen sections and there is a risk that seedlings in the ureter and in the pelvis from retro-peristalsis may be overlooked. It is only justifiable if the other kidney is absent or functioning badly.

PROGNOSIS

Two of the 9 patients are dead. In *Case* 2 the tumour had spread locally, and in *Case* 3 it was sessile and had involved the muscle layers. As for the other 7 the time is too short for any opinion to be offered. The thin ureteric wall and the invasive character of the tumour are the important factors in prognosis. In the low-grade non-invasive papillary type the outlook is good.

SUMMARY

Nine cases of primary ureteric tumour are recorded and their relationship to bladder carcinoma is discussed. Hæmaturia and signs due to ureteric obstruction are the main symptoms. The importance of uretero-pyelography as a method of diagnosis is stressed.

Nephro-ureterectomy with a bladder cuff is the operation of choice provided the other kidney is functioning well.

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THE RESULTS OF TREATMENT OF PORTAL HYPERTENSION

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THERE is still a good deal of controversy and, indeed, some scepticism, regarding the permanent effectiveness of shunt operations, and other surgical procedures, in the treatment of portal hypertension.

During the past eleven years in Newcastle, 55 patients suffering from portal hypertension have been subjected to operation of one form or another. Compared with other published series the number is small, but, nevertheless, it is felt that the experience is enough to warrant certain broad conclusions being drawn.

Table I shows all the operations which have been performed up to the end of January, 1960. With very few exceptions, the indication for operation has been severe, and often repeated, hæmorrhage. I do not at the moment believe that it is justifiable to perform these operations as prophylactic measures in people with known varices but who have not bled.

Table II shows those patients who have died following a porto-caval anastomosis.

Case 1 did well for about three years after operation. She then moved from Newcastle, and we had great difficulty in tracing her. However, we eventually found that she died in November, 1955, of what was said to be liver failure.

Case 2 should probably have been turned down for surgery because of the severity of her cirrhosis. Post-operatively, although the bleeding stopped, she remained a chronic invalid, with jaundice, some ascites, and recurring episodes of protein intoxication. She eventually went into hepatic coma and died at home.

In *Case* 3 the operation was difficult because of the presence of multiple fleshy glands in the gastro-hepatic omentum and a thick-walled atheromatous portal vein. It would probably have been wiser to have abandoned the operation, because the likelihood of the stoma thrombosing under these circumstances is considerable.

Case 4 was first seen in March, 1953, with jaundice, ascites, and recurrent gastro-intestinal bleeding; her liver function tests were grossly abnormal. She was not considered fit for operation at that time, but after five or six months' medical treatment her condition had improved, and the jaundice and ascites had cleared. After operation, however, they both reappeared, and, although she had no further hæmorrhages, she required frequent paracentesis. She eventually died of liver failure eleven months after operation.

Case 5 was making normal post-operative progress but, unfortunately, a week after operation she strangulated a large umbilical hernia. We repaired this, but she then went into hepatic coma and died.

Table I.—PORTAL HYPERTENSION—ALL CASES
(60 operations on 55 patients: 30 patients surviving, 25 died)

OPERATION	INDICATION	NUMBER	RESULT	
			Alive	*Dead*
Porto-caval	Hæmorrhage	29	15	14
Spleno-renal	,,	5	3	2
Splenectomy and gastro-œsophageal devascularization	,,	8	6	2
Splenectomy and gastric transection	,,	6	4	2
Splenectomy and œsophago-gastrectomy	,,	3	1	2
Splenectomy and omentopexy	Ascites.	2	0	2
	Failed spleno-renal	1	0	1
Splenectomy	Hæmorrhage	4	3	1
Ligature of hepatic artery and omentopexy	Ascites	2	0	2

Case 6 remained well following her porto-caval in July, 1954, apart from occasional bouts of indigestion, until August, 1959; she then returned with a further hæmorrhage. A splenic venogram showed a normally-functioning stoma, and at œsophagoscopy no varices could be seen. A barium meal showed some irregularity high up on the lesser curve and a gastroscopy showed what appeared to be a large malignant ulcer encroaching on the cardia. At operation we found a benign ulcer, the size of a saucer, high up on the lesser curve with the main splenic artery in its floor. A total thoraco-abdominal gastrectomy was performed, but this proved too much for her and she died thirty-six hours later. At autopsy, the porto-caval stoma was patent.

Case 7 was found to have very large tortuous collaterals in the gastro-hepatic omentum, replacing the portal vein, which was thrombosed and obliterated. One of these collaterals was used for a side-to-side anastomosis to the inferior vena cava. Post-operatively, he continued to bleed, and, therefore, a fortnight later, splenectomy with high gastric section and re-suture was performed. This again had no effect on the bleeding and he eventually died

on December 25. At autopsy, the stoma was completely thrombosed.

Case 8 should probably have been turned down for operation. She was 67 years old, with severe cirrhosis and various other disabilities as well. She went into coma ten days post-operatively; she recovered temporarily but lapsed back again after a week and died. At autopsy, the stoma was patent.

Case 9 had an episode of severe protein intoxication one month after operation; she recovered with restricted protein and neomycin, and then remained reasonably well on a low protein diet until March, 1959, when she was re-admitted in hepatic coma, from which she died.

Case 10, a 12-year-old boy, had had a splenectomy elsewhere in January, 1951; a spleno-renal shunt was not done because of the disparity in size between the splenic and renal veins. Following the porto-caval in May, 1956, he had two slight bleeds in August of that year, but after this he remained well until November, 1958, when he had further torrential hæmorrhages from which he died. At autopsy, the porto-caval stoma was completely occluded and epithelialized, although at the time of the operation the portal vein had shown no sign of atheroma or thrombosis.

Case 11 developed a mild episode of protein intoxication one month after operation which responded in twenty-four hours to restricted protein and neomycin. When seen in September, 1957, she was well, but she died of liver failure in March, 1958.

Case 12 had severe cirrhosis with jaundice and should probably have been turned down for surgery. She had a further slight hæmorrhage at the end of March, 1959. We had arranged to re-admit her a month later for a repeat splenic venogram but she died at home of bronchopneumonia before we could do this.

Case 13 was in hepatic coma for four days post-operatively and developed ascites; he then recovered but had a further hæmorrhage two months later, again passed into coma, and died. Autopsy showed thrombosis of the stoma.

Case 14, a chronic alcoholic, had a porto-caval shunt in November, 1952. Following this, he had a persistent hypersplenism, and splenectomy was therefore performed in February, 1953. After this, the blood-picture returned to normal. He remained well until May, 1955, when he had a further moderate hæmorrhage followed by an episode of protein intoxication. He recovered from this, but in December, 1959, he had a very severe hæmorrhage from

Table II.—Deaths following Porto-caval Anastomosis

	Sex	Age	Date of Operation	Date of Death	Cause of Death	Remarks
Case 1	F.	64	20.6.52	Nov. 1955	Liver failure	Moderate cirrhosis and gall-stones. Episode of protein intoxication, Aug. 1953
Case 2	F.	45	26.7.52	Jan. 1953	Liver failure	Severe cirrhosis with jaundice. Post-operation: severe protein intoxication
Case 3	F.	42	13.12.52	17.12.52	Liver failure. Rheumatic carditis	Severe cirrhosis; gall-stones; rheumatic carditis with mitral stenosis; portal vein atheromatous. At autopsy, stoma thrombosed
Case 4	F.	52	20.11.53	Oct. 1954	Liver failure	Severe cirrhosis with jaundice and ascites
Case 5	F.	30	6.2.54	20.2.54	Liver failure. Strangulated umbilical hernia	Severe cirrhosis. Making good progress until 1 week post-operatively; then developed strangulated umbilical hernia—repaired; after this, went into hepatic coma
Case 6	F.	52	17.7.54	10.10.59	Liver failure. Gastric ulcer	Moderate cirrhosis. Recurrent hæmorrhage Aug. 1959; stoma patent; large gastric ulcer; total gastrectomy on 8.10.59
Case 7	M.	20	27.11.54	25.12.54	Recurrent hæmorrhage	Severe cirrhosis. Portal vein thrombosed. Large collateral anastomosed to inferior vena cava. Continued to bleed. Splenectomy and gastric section on 9.12.54. Continued to bleed. Post-mortem: stoma thrombosed
Case 8	F.	67	1.10.55	1.11.55	Liver failure. Nephro-sclerosis. Basal collapse	Severe cirrhosis with jaundice. Also essential hypertension, diabetes, and epilepsy. Hepatic coma 10 days post-operatively. Post-mortem: stoma patent
Case 9	F.	53	4.2.56	Mar. 1959	Liver failure	Severe cirrhosis. Severe protein intoxication one month post-operatively
Case 10	M.	12	11.5.56	Nov. 1958	Recurrent hæmorrhage	Severe cirrhosis. Jan. 1951: splenectomy. Further hæmorrhage Aug. 1956 and Nov. 1958. Post-mortem: stoma completely occluded
Case 11	F.	65	5.1.57	31.3.58	Liver failure. Broncho-pneumonia	Mild cirrhosis; gall-stones. Protein intoxication one month post-operatively
Case 12	F.	50	12.7.58	30.4.59	Bronchopneumonia. Liver failure	Severe cirrhosis with jaundice. Slight recurrent hæmorrhage on 21.3.59
Case 13	M.	55	2.8.58	Oct. 1958	Liver failure	Severe alcoholic cirrhosis. Portal vein atheromatous. Recurrent hæmorrhage two months post-operatively. Post-mortem: stoma thrombosed
Case 14	M.	39	8.11.52	Dec. 1959	Hæmorrhage from chronic duodenal ulcer	Moderate alcoholic cirrhosis and hypersplenism; splenectomy 7.2.53. Recurrent hæmorrhage and episode of protein intoxication May, 1955

which he died. Autopsy showed a patent and functioning porto-caval stoma, a few remaining varices, and a large, deeply penetrating, posterior wall duodenal ulcer from which the bleeding had been taking place. The hæmorrhage in 1955 may also have been from this ulcer.

Table III shows those surviving after a porto-caval shunt. Of these 15 patients, 10 made a straightforward recovery and are alive and symptom-free for periods ranging from four months to seven years

Table III.—Patients surviving after Porto-caval Anastomosis

	Sex	Age	Date of Operation	Remarks
Case 1	F.	52	28.2.53	Moderate cirrhosis and follicular reticulosis
Case 2	F.	62	6.3.54	Moderate cirrhosis and gall-stones
Case 3	F.	49	30.10.54	Moderate cirrhosis
Case 4	M.	33	14.5.55	Moderate cirrhosis
Case 5	M.	33	2.7.55	Mild cirrhosis
Case 6	M.	13	12.11.55	Severe cirrhosis. ? nutritional. Preliminary laparotomy and liver biopsy on 25.6.55 followed by severe hæmorrhage and hepatic coma. No complications following porto-caval anastomosis
Case 7	F.	51	8.12.56	Severe cirrhosis with jaundice. Since operation, recurrent episodes of protein intoxication
Case 8	M.	48	2.2.57	Severe alcoholic cirrhosis with temporary ascites
Case 9	M.	33	22.6.57	Moderate cirrhosis. Episode of protein intoxication three weeks post-operatively
Case 10	M.	54	11.1.58	Moderate cirrhosis. Anastomosis appeared satisfactory but 5 days post-operatively further severe hæmorrhage which continued until 28.1.58 when splenectomy and gastric section performed. No further hæmorrhage since
Case 11	M.	32	22.3.58	Moderate cirrhosis
Case 12	F.	12	11.4.59	Congenital hepatic fibrosis. Severe recurrent hæmorrhage on 15.6.59. Splenectomy and devascularization performed. No hæmorrhage since
Case 13	M.	69	10.10.59	Severe cirrhosis. Severe protein intoxication three weeks post-operatively
Case 14	F.	18	9.1.60	Congenital hepatic fibrosis with congenital cystic kidneys
Case 15	F.	45	30.1.60	Severe cirrhosis

after operation. The other 5 have had troubles of one sort or another, usually either recurrent hæmorrhage (in 2 cases) or episodes of protein intoxication (in 3 cases). In the 2 patients with recurrent hæmorrhage the stoma had appeared entirely satisfactory, and there is no obvious explanation of why they should have bled again: following splenectomy and high gastric section and re-suture in one, and splenectomy and gastric devascularization in the other, the bleeding stopped and has not recurred. Of the 3 patients who have had episodes of protein intoxication, in 1 it was relatively slight, occurring on one occasion only, and was fairly easily corrected by a reduced protein intake and a short course of neomycin. In the other 2 patients, the attacks have been severe, have recurred at intervals, and have necessitated a permanent restriction of protein intake to 20–30 g. a day with a small permanent maintenance dose of neomycin. There is no doubt that this is a serious and unpleasant possible sequela of a shunt operation, and it appears especially liable to occur in the more elderly patients with severe cirrhosis; it was encountered in 8 of the patients who have had a porto-caval shunt, an incidence of nearly 30 per cent. It can, of course, occur in any patient with an extensive collateral circulation, but we have not encountered this, nor have we seen it follow any of the other types of operation that have been performed. In a patient over 50 years of age with severe cirrhosis, especially if there is an abnormal pre-operative electro-encephalogram, it is probably wiser to undertake some form of direct attack on the varices rather than do a shunt operation. There is no doubt that the patients who do best following a shunt are those under 50 years of age, and who have mild or moderate cirrhosis. It has been said, of course, that this group might do just as well with conservative medical treatment, but it is difficult to believe that the cessation of hæmorrhage following operation, and its failure to recur for periods ranging up to seven years in this series, is purely a matter of coincidence or chance.

Table IV shows the results in 5 cases of end-to-end spleno-renal anastomosis, performed after removal of the spleen and left kidney. These were the first cases done in the series, and because of personal dissatisfaction with the operation, and doubts about its permanent efficacy, it has not been persisted with. Other surgeons, however, have claimed good results with it, and it may be that, in spite of its drawbacks, it is still the best operation for the difficult group of patients, especially children, with extra-hepatic obstruction.

Of the 5 patients, 2 died a few days after operation, 1 of bilateral pulmonary collapse and 1 of thrombosis of the splenic vein with infarction of the stomach. In this patient, at autopsy, the stoma was completely occluded by thrombus, but in the other it was patent.

Of the 3 patients who have survived, 2 have had further hæmorrhages, and in them the stoma is

almost certainly occluded. We explored the portal vein in one of them and found it completely thrombosed. As is well known, there is a fairly high incidence of thrombosis of the portal vein following splenectomy. We have had 4 patients who had had a previous splenectomy and whose portal veins we subsequently explored; in 3 of them the vein was thrombosed. Splenectomy should therefore be avoided, if possible, with patients of whom it is performed through a left thoraco-abdominal incision. There have been no permanent ill-effects in any of our patients as a result of this extensive interference with the blood-supply of the stomach and œsophagus.

Of the 8 patients, 2 have died, one a fortnight after operation, and the other seven months after operation; six survive, although in 2 of them there have been recurrent slight hæmorrhages. One of the patients who died was a fat, elderly female of 69 years,

Table IV.—SPLENO-RENAL ANASTOMOSIS

	Sex	Age	Date of Operation	Result	Remarks
Case 1	M.	34	31.3.49	Alive	Mild cirrhosis. Slight recurrent hæmorrhage 1950 and 1952
Case 2	M.	36	4.8.49	Dead	Died 6 days post-operatively. Post-mortem: Thrombosis of stoma and splenic vein with infarction of stomach
Case 3	M.	31	20.1.50	Dead	Died 3 days post-operatively. Post-mortem: Bilateral pulmonary collapse. Stoma patent
Case 4	F.	24	29.4.50	Alive	Mild cirrhosis
Case 5	M.	38	24.5.51	Alive	Mild cirrhosis. Recurrent hæmorrhage Aug. 1956. Portal vein explored—thrombosed. No further hæmorrhage since

Table V.—SPLENECTOMY AND GASTRO-ŒSOPHAGEAL DEVASCULARIZATION

	Sex	Age	Date of Operation	Indication	Result	Remarks
Case 1	F.	27	13.7.57	Splenic vein thrombosis	Alive	Slight recurrent hæmorrhage, Aug. 1958
Case 2	M.	34	1.3.58	Portal vein thrombosis	Alive	Slight recurrent hæmorrhage, Aug. 1958
Case 3	M.	12	10.12.58	Splenic vein thrombosis	Alive	
Case 4	M.	50	13.6.59	Thrombosis of portal vein following previous splenectomy	Dead, 26.1.60 recurrent hæmorrhage	Several episodes of recurrent hæmorrhage since operation
Case 5	F.	69	16.6.59	Emergency operation for control of hæmorrhage	Dead	Fat, elderly patient with severe cirrhosis. Died 2.7.59. Post-mortem: Pulmonary infarcts
Case 6 (Case 12, *Table III*)	F.	12	16.6.59	Recurrent hæmorrhage after porto-caval anastomosis on 11.4.59	Alive	Congenital hepatic fibrosis
Case 7	M.	6	25.9.59	Emergency operation for control of hæmorrhage	Alive	Congenital hepatic fibrosis
Case 8	M.	53	28.11.59	Moderate cirrhosis with calcified portal vein	Alive	

thought a porto-caval shunt may subsequently prove necessary. The fifth patient has been well and symptom-free for ten years.

Table V shows the results in 8 patients who have had a splenectomy and gastro-œsophageal devascularization. The rationale of this, and similar procedures, is to try to reduce the volume of blood-flow through the varices, and the pressure within them, by disconnecting them from their portal blood-supply, while at the same time retaining as many of the external collaterals as possible. The operation does not reduce the portal pressure, if this is raised, and, in theory at any rate, one would not expect it to be particularly effective. However, in practice the results have been reasonably encouraging. The operation, which can be tedious and difficult, consists in freeing the upper two-thirds of the stomach and the lower 3 in. of œsophagus of all their external vascular connexions, and it can only be satisfactorily with poor liver function tests, and we were not keen to operate on her at all. Following operation, she had no further hæmorrhages, but her condition slowly deteriorated: she became jaundiced, and died on the sixteenth day. The other patient who died also had cirrhosis. An emergency splenectomy for hæmorrhage had been performed elsewhere in January, 1959; exploration of the portal vein in March, 1959, showed it to be thrombosed. Following devascularization in June, he had a further hæmorrhage in October, and another in January, 1960, from which he died.

The last patient in this group, although he only had a moderate cirrhosis, had a gross calcification of the portal vein, and this, of course, precluded a shunt operation.

The two most interesting patients in this group are, perhaps, the two children, who had congenital hepatic fibrosis. In the case of the 12-year-old girl,

a porto-caval anastomosis was performed on April 11, 1959; the operation was straightforward and one anticipated a good result. However, two months later, there was recurrent very severe bleeding and an emergency splenectomy and devascularization was performed; the child was too ill for us to do a venogram on this occasion. There have been no further hæmorrhages since this second operation. The other child was also operated on as an emergency for severe bleeding. A few weeks earlier, this boy's sister, aged 9 years, had been admitted with exactly the same condition. She died in the anæsthetic room following a massive hæmorrhage while being prepared for operation a few hours after admission. The boy has had no further hæmorrhages since operation.

An alternative to splenectomy and devascularization is splenectomy with high gastric section and re-suture with a more limited gastric devascularization. The principle is the same, but the aim is not only to interrupt the extrinsic portal supply to the varices but also to divide the vessels running in the gastric wall. We have not been very convinced, however, that this additional step does a great deal to diminish the blood-flow to the varices; moreover, in one case, a patient who had had this operation two years later developed a large gastric ulcer at the site of gastric section. This required a total gastrectomy from which he recovered, but he died seven months later from liver failure. *Table VI* shows the 6 patients who have undergone this operation. The other patient in this group who died has already been mentioned in the porto-caval group. The 4 surviving patients are in good health, although 1 had a small recurrent hæmorrhage six months after operation. The man who bled after an apparently satisfactory porto-caval anastomosis has already been mentioned; he has not bled since the second operation.

There is a small group of 3 patients who have had a splenectomy and œsophago-gastrectomy (*Table VII*); the last 2 of these have already been mentioned.

Table VI.—Splenectomy and Gastric Section

	Sex	Age	Date of Operation	Indication	Result	Remarks
Case 1	F.	59	22.5.54	Mild cirrhosis with thrombosed portal vein	Alive	
Case 2 (Case 7, *Table II*)	M.	20	9.12.54	Recurrent hæmorrhage after anastomosis of collateral to inferior vena cava on 27.11.54	Dead	Hæmorrhage continued after 2nd operation. Post-mortem: stoma thrombosed
Case 3	M.	27	5.3.55	Severe cirrhosis with thrombosed portal vein	Dead	Recurrent hæmorrhage Dec. 1957. Large gastric ulcer and persistent varices; total gastrectomy. Died July, 1958; liver failure
Case 4	F.	59	28.9.57	Mild cirrhosis	Alive	Two previous operations on gall-bladder and common bile-duct; massive adhesions around portal vein. Slight recurrent hæmorrhage, March, 1958
Case 5 (Case 10, *Table III*)	M.	54	28.1.58	Recurrent hæmorrhage after porto-caval anastomosis on 11.1.58	Alive	
Case 6	F.	13	24.5.58	Portal vein thrombosis	Alive	

Table VII.—Splenectomy and Œsophago-gastrectomy

	Sex	Age	Date of Operation	Indication	Result	Remarks
Case 1	M.	6	22.1.53	Portal vein thrombosis	Alive	Post-operative œsophageal stricture
Case 2 (Case 3, *Table VI*)	M.	27	28.12.57	Large gastric ulcer and persistent varices following gastric section	Dead	Died July 1958—liver failure
Case 3 (Case 6, *Table II*)	F.	52	8.10.59	Large gastric ulcer following porto-caval anastomosis on 17.7.54	Dead	Died 10.10.59—liver failure

In the boy of 6 years, with an extra-hepatic obstruction, we removed the pile-bearing area in the lower œsophagus and along the lesser curve, closed the stomach, and anastomosed the œsophagus to the fundus in the posterior mediastinum. He subsequently developed a stricture at the stoma, but this has slowly improved and he has remained in fairly good health until three or four months ago when he developed a secondary anæmia due to a fairly severe œsophagitis; there is no evidence now of recurrent varices or of stricture.

Theoretically, this operation would appear to be the most rational and effective alternative to a splenorenal anastomosis in the treatment of extra-hepatic obstructions. Its great drawback is the destruction of the cardiac sphincter mechanism and the subsequent risk of œsophagitis and of œsophageal stricture.

There are, finally, three small groups of miscellaneous procedures which can be dismissed fairly briefly. The operations for ascites were done several

years ago, and one would not now operate on this type of patient. Ligature of the hepatic artery was a disastrous procedure; both patients on whom it was done died of acute liver necrosis a few days after operation. Four patients had splenectomy only: the first, a young man of 26 years, had a primary splenic vein thrombosis; the second, a man of 31 years, had a mild cirrhosis, but, in addition, in the past he had had a tuberculous spine and a tuberculous peritonitis which had left dense adhesions in the upper abdomen; these made mobilization of the portal vein impossible. In both these patients, splenectomy was accompanied by severe hæmorrhage, and we did not think it wise to do anything more. Both are still alive, five and five and a half years respectively after operation; the first man has had two or three episodes of moderately severe recurrent hæmorrhage, the second has had no further hæmorrhages. The third patient was a man of 62 years with severe alcoholic cirrhosis who had had a moderately severe hæmorrhage but who also had a marked degree of hypersplenism. He was a very unfit patient, and although he just managed to survive a splenectomy, it is doubtful if he would have stood anything more. The fourth patient in this group, the one who died, was a young man of 31 years with mild cirrhosis. He was advised to have a porto-caval, but refused, and would only agree to a splenectomy. This was therefore done. Two and a half years later, he returned with further severe hæmorrhages; on this occasion, he agreed to a porto-caval, but, on exploration, the portal vein was found to be thrombosed. He continued to bleed after operation, and died.

The two splenectomies in which we encountered severe hæmorrhage were done by an abdominal approach, and this is a mistake; these large vascular spleens are far better dealt with by a thoraco-abdominal approach, and this approach is, of course, essential if a devascularization procedure is to be done as well.

CONCLUSIONS

A porto-caval anastomosis should be considered in those patients, especially under the age of 50 years, who have mild or moderately severe cirrhosis, and who have bled. In severe cirrhosis, especially in patients over the age of 50 years, one has to balance the risks of further hæmorrhage, if operation is not done, against the possible risks of cerebral disturbances, sometimes severe, if a shunt is done. In this type of patient, a splenectomy and devascularization, or other form of direct attack on the varices, has a great deal to commend it; not the least of its advantages is that it can be undertaken by surgeons who may feel disinclined to tackle venous shunt operations.

The second main conclusion is that, although many procedures have been tried for cases of extra-hepatic obstruction, none of them is entirely satisfactory, and this is a pity, because many of the patients in this group are, of course, children and young adults.

Acknowledgements.—I should like to express my gratitude to Sir James Learmonth and to Dr. H. A. Dewar, who first stimulated my interest in the treatment of this condition; to Professor John Gilmour and Mr. A. Hedley Whyte for their unfailing interest and help; to Dr. C. K. Warrick, who has done most of the radiology; and to Dr. E. I. Tate, who has given all the anæsthetics. I should like also to thank all those members of the medical and nursing staff of the Royal Victoria Infirmary, Newcastle upon Tyne, too numerous to mention, who have spared no effort in the pre- and post-operative care of these difficult cases.

SPONTANEOUS INTRAPERITONEAL RUPTURE OF THE URINARY BLADDER

By CYRIL O. INNIS, North Borneo

Spontaneous rupture of the urinary bladder, defined as rupture of the bladder occurring without any history of trauma, is a rare condition. Bastable, De Jode, and Warren (1959) state an incidence of 1 in 126,000. Recently, cases have been reported by Yarwood (1959), Thomas (1956), and Graves (1960). The mortality-rate from this condition is high. Stone (1931) gives a rate of 45·4 per cent, Bacon (1943) 44·2 per cent, and Bastable and others (1959) 47 per cent.

The following case is reported as it presents one or two interesting features.

CASE REPORT

The patient, a male aged 20 years, a member of one of the Dusan tribes of Borneo, was admitted to the Duchess of Kent Hospital, Sandakan, on Nov. 25, 1959, with the following history:—

Chief complaints: (1) Inability to pass urine for 5 days; (2) Abdominal pain for 5 days; (3) Vomiting for 2 days.

History of Present Illness.—Five days prior to admission into hospital the patient found that he was unable to pass urine. There was no sudden stoppage of the stream of micturition, the patient being unable to void urine although desiring to do so. This was followed by pain of gradually increasing intensity over the suprapubic region, associated with a swelling in the same area. The swelling gradually increased in size and extended up to the umbilicus. Three days after the onset of the above symptoms the patient had a strong desire to defæcate and while attempting to do so he experienced sudden intense pain over the suprapubic region which soon became generalized to the whole abdomen. Twenty-four hours later the patient started vomiting after taking food. The vomitus was brownish coloured fluid copious in amount and he vomited five times in the twenty-four hours prior to admission. The patient denied any history of trauma on close and direct questioning.

Past History.—The patient had been treated for tuberculosis of the lung and right elbow-joint six months previously. At the time of admission to hospital the lesion of the elbow-joint had healed and there was no active disease of the lung. While undergoing treatment

for these two conditions the patient had an attack of renal colic. This was investigated and an intravenous pyelogram proved negative.

EXAMINATION.—The patient was in great distress. General condition was fair, tongue dry and coated. B.P. 158/110. Pulse 100.

Abdomen.—

1. There was generalized distension of the abdomen with bulging of both flanks.

2. Umbilicus was centrally situated and was not everted.

3. Generalized rigidity with tenderness most marked in the suprapubic region.

FIG. 181.—Photograph taken at the time of operation to show extent of tear in bladder wall.

4. Dullness of abdomen on percussion and a fluid thrill was elicited.

5. Bowel sounds could not be heard.

6. Rectal examination showed bogginess, and tenderness of the rectum.

Genitalia dribbling of clear urine from the penile urethra but no evidence of extravasation of urine.

Nervous System.—N.A.D.

A diagnosis of intraperitoneal rupture of the bladder was made in the above findings. An intravenous infusion of normal saline was started and the patient was prepared for immediate laparotomy.

OPERATION.—Under a general anæsthetic (open ether) catheterization with a metal catheter was attempted but failed. A definite obstruction to the entry of the catheter into the bladder could be felt.

The bladder was exposed through a midline infra-umbilical incision and opened. An exploring finger revealed a loop of small bowel and a stone about the size of the fifth fingernail impacted in the internal meatus. The stone was removed. The peritoneal cavity was opened. This was found to contain a pale yellow fluid with an odour of urine. One hundred and sixty ounces of this fluid were aspirated; a fair quantity, in addition, overflowed on to the table and floor. The bowel was found to be injected but peristalsis was visible. The tear in the bladder was situated on the posterosuperior surface extending longitudinally from the prostate and was approximately $\frac{1}{2}$–$\frac{3}{4}$ in. long; the edges were friable. There was no macroscopic evidence of disease of the bladder wall.

The bladder wall was repaired in three layers with interrupted catgut sutures. The peritoneal cavity was mopped dry and closed with drainage. A suprapubic cystostomy completed the procedure.

A specimen of fluid removed from the abdomen did not reveal the presence of pus cells or organisms.

Post-operatively antibiotics were given in large doses and intravenous fluids continued for 48 hours. His recovery was uneventful. The suprapubic drain was removed on the 15th day when it was found that the patient could void urine normally. On the 14th post-operative day bougies were passed but no sign of a stricture found.

The patient absconded from hospital on Dec. 29, before a post-operative cystoscopy could be done. I thought this was the last I would see of him, as few people from the interior tribes return to follow-up clinics, but to my surprise, six months later, while on a medical tour of the interior, he volunteered his services as a 'paddler' for my canoe. He was symptom-free and voiding urine normally but no amount of persuasion would induce him to return for further investigation.

DISCUSSION

In searching the recent literature I have been able to find 1 case reported by Stone (1931) where rupture could possibly have been attributed to a calculus. In the series presented by Bastable and others (1959) one other case is recorded. In the case described above, a stone was impacted in the internal meatus and rupture followed on an increase in intra-abdominal pressure during an attempt to defæcate.

Owing to the delay in seeking treatment this case presented a classical clinical picture and the diagnosis was not in doubt. The period of time which elapsed between the symptoms of rupture and surgical intervention estimated at 48 hours is, I feel, worthy of note. The fact that the urine was sterile was probably an important factor contributing to a successful outcome. In Bastable and others' (1959) series a higher mortality-rate was found in cases where rupture was associated with tuberculosis or carcinoma of the bladder and enlargement of the prostate. In this case, although tuberculosis of the bladder was not excluded by biopsy, there was no naked-eye evidence of disease and this was looked for in view of the known history of tuberculosis. It would appear that factors favouring a good prognosis are: (1) Absence of urinary infection; (2) Absence of associated disease of the bladder. Prompt laparotomy with repair of the rupture and drainage of the peritoneal cavity has also been emphasized. Although I am in full agreement with this as a sound surgical principle, this factor could hardly be said to have operated in this case.

SUMMARY

A case of intraperitoneal rupture of the urinary bladder is presented. Recovery followed surgical intervention 48 hours after the onset of rupture.

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NECROTIZING COLITIS

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Gangrene of the colon was first described by Lauenstein in 1882. It is rare, and strangulation in a hernia, volvulus, or intussusception is its most usual cause. Mesenteric arterial or venous occlusion in the colic vessels may also cause gangrene of the colon, but much less commonly than in the small bowel.

This paper reports 6 cases of a gangrenous process affecting the colon in which there was an extensive necrosis of the mucous membrane with patches of full-thickness gangrene of the bowel wall. The patients were admitted to Central Middlesex, St. Mark's, and Westminster Hospitals between September, 1957, and February, 1960. The clinical and pathological features in all were the same. We believe the description of this group as a disease entity specifically involving the colon has not been reported hitherto.

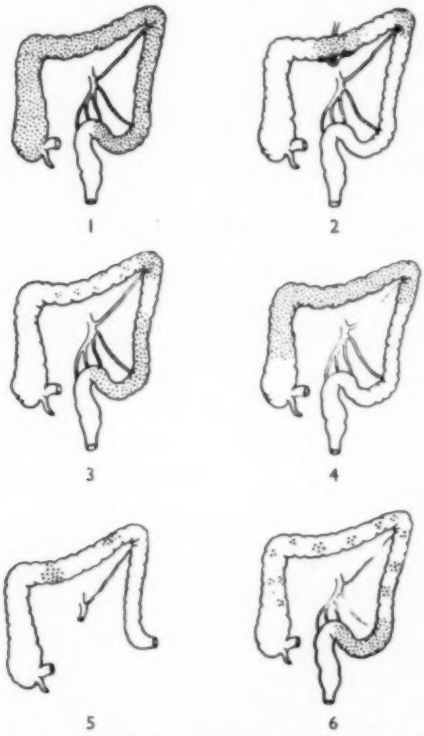

Fig. 182.—Showing the distribution of the gangrene in the six cases as seen on the serosal aspect.

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† Now at The Middlesex Hospital, London.

CASE REPORTS

Case 1.—F. H., a woman aged 65 years, was admitted to Central Middlesex Hospital on August 6, 1957. She wakened at 7 a.m. on the day of admission with severe abdominal pain which commenced in the left iliac fossa and quickly became generalized. It was severe, constant, and accompanied by vomiting. The patient passed a normal stool one hour after the onset of the pain. She had been investigated in 1955 for epigastric pain and a barium meal had shown a diverticulum in the third part of the duodenum. For the year previous to her admission she had been very well.

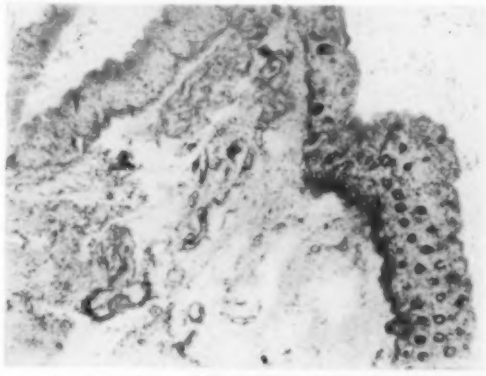

Fig. 183.—*Case* 1. Showing necrosis affecting the mucosa with a gradation from partial to complete necrosis. Marked submucosal œdema is also seen. H. and E. (× 30.)

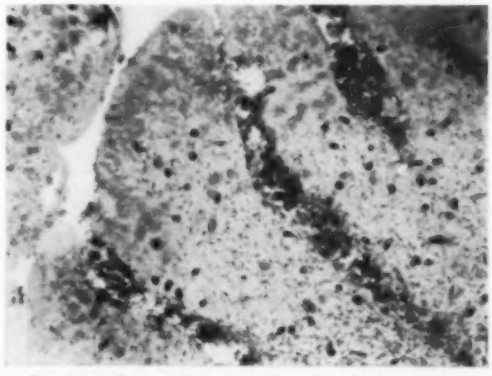

Fig. 184.—*Case* 1. Large numbers of Gram-positive rod-shaped organisms are shown in the mucosa. Gram. (× 313.)

On Examination.—She was thin and was obviously in severe pain. Her temperature was normal, pulse-rate 86 and blood-pressure 120/70 mm. Hg. The abdomen was distended with generalized tenderness and rigidity. There was evidence of free fluid in the abdomen and a purple discoloration was noticed over the right loin. Rectal examination revealed small hard masses of fæces.

A provisional diagnosis of perforated diverticulitis of the sigmoid colon was made.

LAPAROTOMY was undertaken the same day and on opening the peritoneal cavity a large amount of dark fluid presented. Gangrene of the large bowel was found extending from the cæcum to the mid-sigmoid region (*Fig.* 182). There were petechiæ over the distal sigmoid colon and rectum. Examination of the colic arteries showed some of them to pulsate, but the majority were pulseless. The veins were seen to be engorged.

FIG. 185.—*Case* 2. Complete necrosis of the mucosa has occurred and a most intense polymorphonuclear infiltration of the submucosa is present. H. and E. (× 30.)

Resection of the gangrenous bowel was carried out and the ileum and distal sigmoid colon were both brought through a grid-iron incision in the left iliac fossa as an ileostomy and colostomy.

The specimen measured 110 cm. in length. The bowel wall was extremely thin. The lumen did not contain blood-stained fluid. There was a fibrous stricture 25 cm. from the distal end.

HISTOLOGY.—Examination (*Fig.* 183) showed that many parts of the mucous membrane were necrotic and some areas showed complete death of tissue with intense neutrophil infiltration of the submucosa. In this section there was one thrombosed vein in the submucosa surrounded by acute inflammatory cells and the appearance suggested that the thrombosis was secondary to phlebitis. The muscular coats were mainly intact. The peritoneum showed some inflammatory reaction. Gram staining the histological material revealed numerous colonies of Gram-positive rods (*Fig.* 184). They were present in all layers of the bowel wall but mainly in the mucosa.

POST-OPERATIVE COURSE.—A paralytic ileus developed, and soon after this the edges of the colostomy became gangrenous. Nine days after the operation laparotomy was undertaken for acute intestinal obstruction and this was found to be due to adhesions. Corrective surgery was attempted but the patient continued to deteriorate and died the following day.

Case 2.—A. F., a woman aged 56 years, was admitted to Central Middlesex Hospital on March 12, 1959, complaining of severe abdominal pain which had wakened her at 2 a.m. that morning. The pain was constant, situated initially in the left iliac fossa, and had rapidly become generalized. This was accompanied by vomiting of small quantities of bile. Following the onset of the pain there had been no bowel action. She had complained of weakness, feeling the cold, constipation, and hoarseness for 2 years, and recently had complained of abdominal pain and distension after meals.

ON EXAMINATION.—The patient looked gravely ill and collapsed with a feeble pulse of 100. Blood-pressure was 85/65 mm. Hg. Peripheral cyanosis was very marked. The abdomen was grossly distended, tenderness and rigidity were generalized, and bowel sounds were absent. A provisional diagnosis of generalized peritonitis due to a perforated diverticulitis of the sigmoid colon was made.

LAPAROTOMY.—At operation soon after admission a large amount of serous fluid was found. The most striking feature was the enormous dilatation of the transverse colon. Scattered over the transverse colon were irregular

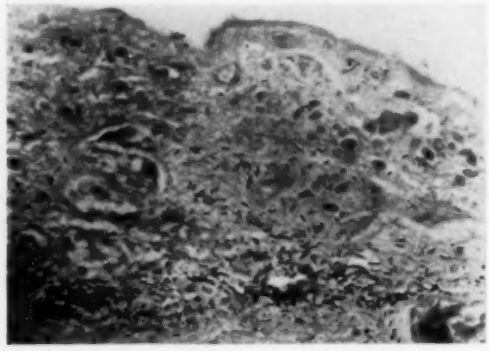

FIG. 186.—*Case* 2. This shows large numbers of Gram-positive organisms in the deeper parts of the mucosa. Gram. (× 313.)

mottled patches of necrosis, grey-green in colour; the splenic flexure was frankly gangrenous. The affected areas (*Fig.* 182) were paper-thin, but the bowel wall and mesocolon were markedly œdematous. The middle colic and left colic arteries were examined and found to be pulsating. The descending colon and sigmoid colon contained hard fæces.

The transverse colon and the descending colon were excised and a proximal right transverse colostomy and distal left iliac colostomy were made. The patient's condition was critical at the end of the operation. Resuscitation was attempted, but the patient did not respond and died before leaving the operating theatre.

Examination of the mucosa showed an extensive necrosis affecting a wide area. The mucosa was black and œdematous but there was no ulceration. Two small polypi were present.

HISTOLOGY.—Examination (*Fig.* 185) showed extensive necrosis affecting mainly the mucosa. In other areas there was full-thickness necrosis of the bowel wall. There was a most intense inflammatory response in the mucosa with many polymorphonuclear leucocytes. One of the polypi was examined and this was a benign adenoma. One small vein in the mesentery contained a thrombus but all other vessels examined showed no abnormality. Gram staining of the section revealed large colonies of Gram-positive rods in all layers of the bowel wall, principally the deeper parts of the mucosa (*Fig.* 186).

AUTOPSY showed atrophy of the thyroid gland. The mesenteric vessels were carefully examined and found to be patent and free of atheroma.

Case 3.—M. F., a woman aged 78 years, was admitted to Central Middlesex Hospital on July 11, 1959, complaining of severe abdominal pain which had commenced suddenly as the patient arose from her bed that morning. The pain was central, remaining localized. It was severe and constant. She vomited several times. There was complete constipation following the onset of the pain and no flatus was passed per rectum. Prior to this illness the patient had enjoyed excellent health.

On Examination.—The patient looked very ill. Her face was pale and contorted with pain. There was peripheral cyanosis in the limbs and also in both loins. The pulse-rate was 95 and the blood-pressure 90/60 mm. Hg. The abdomen was very distended and there was generalized tenderness. Rigidity was absent. Occasional weak bowel sounds were audible. The patient was resuscitated with blood transfusions, and an X ray of the abdomen was taken (*Fig.* 187) showing extreme distension of the large bowel. A provisional diagnosis of volvulus of the sigmoid colon or an intra-abdominal vascular catastrophe was made.

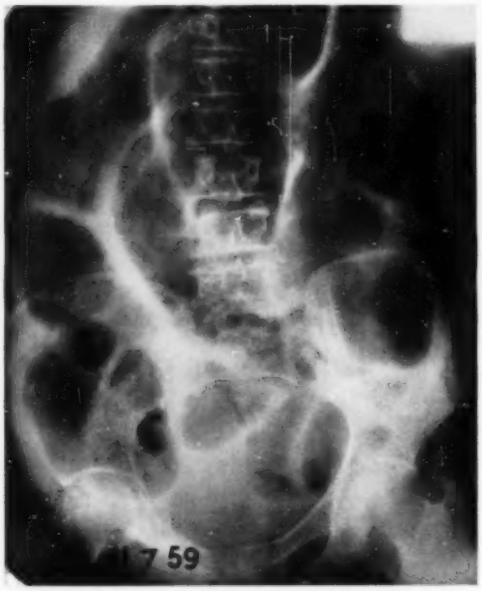

Fig. 187.—*Case* 3. A pre-operative radiograph of the abdomen showing the enormous dilatation of the large bowel.

Laparotomy was performed the same evening and on opening the peritoneum a large quantity of foul-smelling dark green fluid gushed into the wound. The colon was enormously dilated from the hepatic flexure to the recto-sigmoid junction. There were patches of green gangrene of varying size distributed over the bowel (*Fig.* 188). The bowel wall was congested and œdematous. The inferior mesenteric artery was felt pulsating vigorously, but no pulsation was felt in the marginal vessels situated in the œdematous mesentery of the sigmoid colon. The transverse colon, descending colon, and proximal half of the sigmoid colon were excised and an end-to-end anastomosis carried out.

Histology.—Examination (*Fig.* 189) showed marked thinning of the bowel wall and this was thought to be due to the extreme distension. There was complete necrosis of the mucosa and in some areas the muscle coats were similarly affected. There was a patchy infiltration of polymorphonuclear leucocytes. The vessels were engorged and dilated but there was no evidence of vascular disease or thrombosis. Gram staining showed large numbers of Gram-positive rods in the mucosa and submucosa (*Fig.* 190).

Fig. 188.—*Case* 3. This shows the patchy distribution of the necrosis and the enormous dilatation of the upper part of the sigmoid colon which suggested a volvulus when the abdomen was first opened.

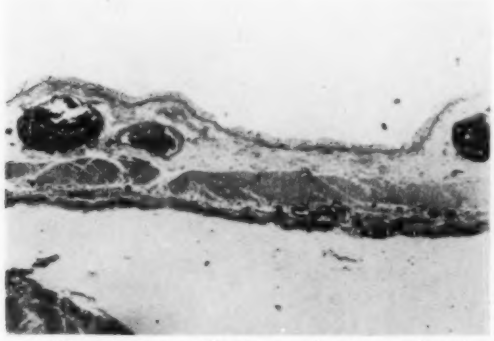

Fig. 189.—*Case* 3. Showing very thin bowel wall with complete necrosis of the mucosa and the dilated vessels in the submucosa. These appearances emphasize the marked distension of the bowel seen at operation. H. and E. (× 30.)

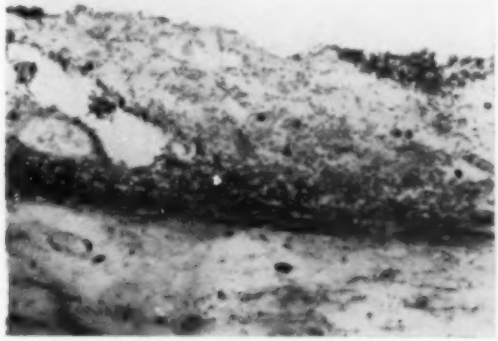

Fig. 190.—*Case* 3. Vast numbers of rod-shaped organisms are seen in the mucosa with many more in the submucosa. Gram. (× 313.)

Post-operative Course.—Following operation the patient initially made a good recovery. During the second

week of the post-operative period she developed intestinal obstruction which settled with intragastric aspiration and intravenous fluids. During the third week a pyrexia developed but resolved when a fæcal discharge presented along the drain wound associated with the anastomosis. The fæcal fistula rapidly closed and her recovery was then uneventful. She was discharged six weeks after admission.

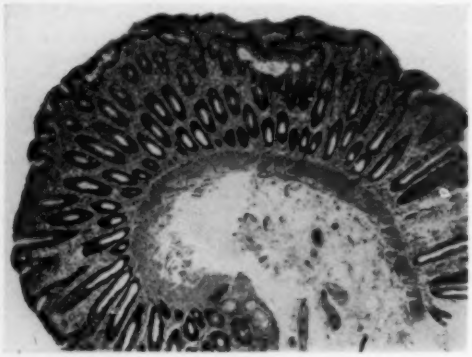

FIG. 191.—*Case* 3. This biopsy of the colon at 20 cm. was taken 9 months after resection of the gangrenous bowel. The mucosa is normal. There is no underlying disease. H. and E. (× 30.)

This patient has been followed up 9 months later. She is very well and has no disturbance of bowel habit. A barium enema shows no abnormality. Sigmoidoscopy carried out to 20 cm. is normal and a mucosal biopsy at 20 cm. shows normal mucosa (*Fig.* 191).

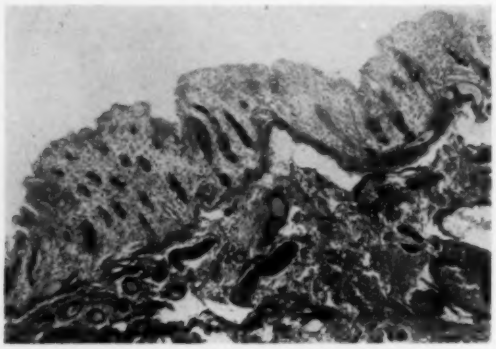

FIG. 192.—*Case* 4. The mucosa is necrotic and the vessels in the submucosa are dilated. Many polymorphonuclear leucocytes are present. H. and E. (× 30.)

Case 4.—E. B., a woman aged 71 years, was admitted to Central Middlesex Hospital on September 13, 1959, complaining of severe abdominal pain. The pain was central and had commenced suddenly. It was accompanied by vomiting. She had been constipated for 3 days prior to the onset of this illness, but previously had enjoyed good health.

ON EXAMINATION.—The patient was obese. She was very distressed and there was peripheral cyanosis. The pulse-rate was 120 and the blood-pressure was 130/80 mm. Hg. The abdomen was distended with generalized tenderness and guarding. Bowel sounds were absent. A provisional diagnosis of generalized peritonitis due to diverticulitis of the sigmoid colon was made.

LAPAROTOMY.—A large quantity of dirty brown offensive fluid was found in the peritoneal cavity. The colon was distended and extensively affected by grey areas of gangrene (*Fig.* 182). The middle colic artery could not be felt pulsating. The gangrenous bowel was excised and an end-to-end anastomosis carried out.

Examination of the mucosa revealed a black, œdematous, and non-ulcerated surface. The colour contrast between serosa and mucosa was very striking.

HISTOLOGY.—Examination (*Fig.* 192) showed necrosis of the mucosa and muscle layers, the changes being most marked in the mucosa. There was an extensive infiltration with polymorphonuclear leucocytes. The vessels were dilated but otherwise normal. Some simple adenomatous polypi were seen. Gram staining showed large numbers of Gram-positive rods within the submucosa and *Fig.* 193 shows them within submucosal veins.

POST-OPERATIVE COURSE.—Following operation the patient remained ill with a rapid pulse of 120 and cold, blue extremities. Six days after operation she complained of severe left-sided abdominal pain and became collapsed with marked cyanosis. The following day a fæcal fistula appeared through the drain wound. Ten days after operation she collapsed suddenly and died. Autopsy showed an extensive peritonitis associated with sloughing of the bowel wall at the anastomosis. The mesenteric vessels were free of atheroma and no thrombosis could be demonstrated.

Case 5.—A. V., a woman aged 69 years, was admitted to St. Mark's Hospital on November 11, 1959, as an emergency, complaining of severe abdominal pain of 4 hours' duration. Previously, in 1954, she had undergone a perineo-abdominal excision of the rectum for an adenocarcinoma. She had remained very well until 3 weeks prior to her admission when the colostomy, which had been functioning regularly, had become constipated. The abdominal pain was severe and central

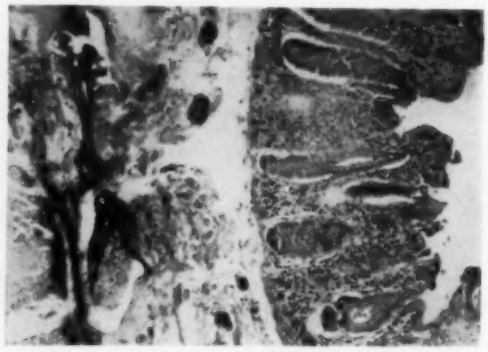

FIG. 193.—*Case* 4. Large numbers of Gram-positive rod-shaped organisms are within the submucosal veins. Gram. (× 80.)

and was accompanied by vomiting. The colostomy had not worked before coming to hospital, but later that day a small amount of blood-stained fluid presented at the colostomy.

ON EXAMINATION.—The patient appeared very ill, with a pale face, and cold, cyanotic extremities. The pulse-rate was 80 and the blood-pressure fell from 140/85 mm. Hg to 110/80 mm. Hg before operation. There was central abdominal tenderness associated with rigidity. There was no distension and bowel sounds were present. A provisional diagnosis of peritonitis associated with a strangulated intestinal obstruction was made.

LAPAROTOMY.—This was undertaken on the day of admission and a large quantity of free, turbid fluid

was found. The transverse and descending colon were intensely congested and œdematous and there were scattered areas of full-thickness gangrene (*Fig.* 194) which felt like wet blotting paper. There was an offensive odour of putrefaction. The mesenteric vessels were carefully examined and were found to be pulsating forcibly.

The colon was transected proximally through the mid-transverse colon and distally 2 in. short of the previous colostomy. The proximal colon was brought out as a colostomy and the distal colon was closed as a blind stump. After the wound was closed the clamp was taken off the exteriorized transverse colon to construct the colostomy. The mucosa of this bowel was œdematous and black and more colon was prolapsed until healthy mucosa was obtained. Resection of this diseased colon (*Fig.* 195) was then carried out and a right transverse colostomy made.

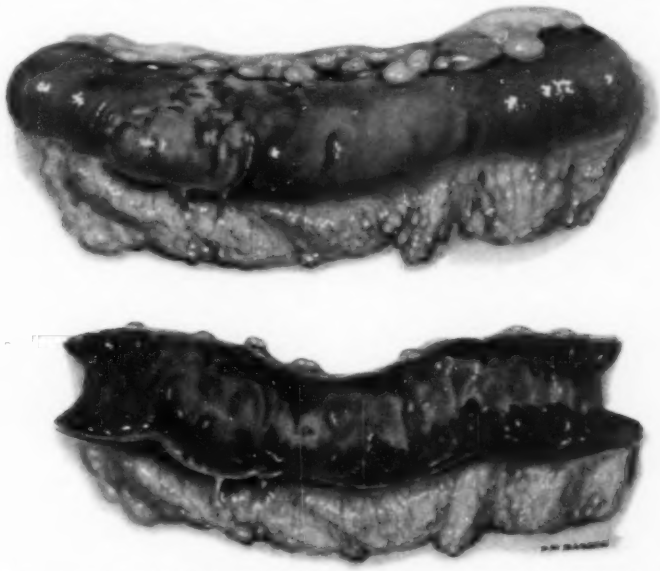

FIG. 194.—*Case* 5. Showing the necrosis on the serosal aspect of the colon and the striking contrast of the mucosal aspect.

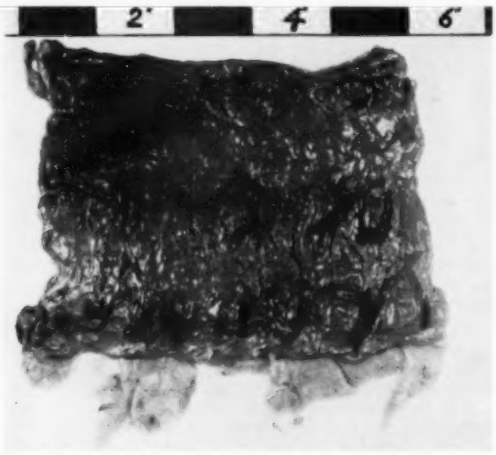

FIG. 195.—*Case* 5. This specimen was removed when the mucosa of the initial colostomy was found to be black. The mucous membrane is shiny, swollen, and non-ulcerated.

FIG. 196.—*Case* 5. The mucous membrane is necrotic. The submucosa is infiltrated with polymorphonuclear leucocytes and œdema. H. and E. (× 30.)

Examination of the resected specimens showed identical changes in the mucosa which was black-brown in colour and œdematous. There was no blood-stained fluid or pus in the lumen. There was no ulceration.

HISTOLOGY. — Histological examination (*Fig.* 196) showed extensive necrosis of the mucosa with almost complete loss of structural detail. The submucosa and muscle layers were congested, œdematous, and a polymorphonuclear infiltration was present. Blood-vessels were distended but no vascular pathology was demonstrated. Gram staining (*Fig.* 197) showed large colonies

of Gram-positive rods which had colonized almost entirely within the submucosa.

POST-OPERATIVE COURSE.—Following operation the patient made a good recovery until she developed a bronchopneumonia in the third week. This responded readily to treatment and she was discharged seven weeks after admission.

FIG. 197.—*Case* 5. Large numbers of Gram-positive rods can be seen colonized almost entirely within the mucosa. Gram. (× 46.)

FIG. 198.—*Case* 6. This shows the mucosal surface. The specimen has been decolorized by formalin but the smooth surface is readily seen.

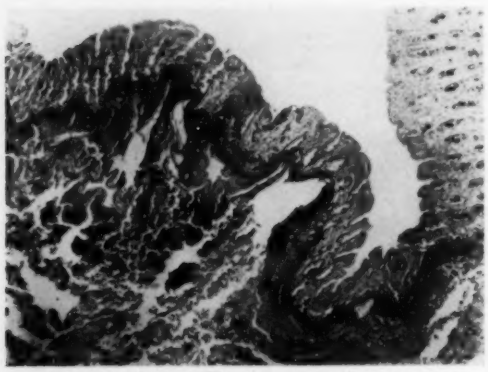

FIG. 199.—*Case* 6. This shows mucosal necrosis, submucosal œdema, and infiltration with polymorphonuclear leucocytes. H. and E. (× 30.)

FIG. 200.—*Case* 6. The Gram-positive organisms are situated almost entirely within the submucosa. Gram. (× 80.)

Case 6.—H. H., a man aged 59 years, was admitted to the Westminster Hospital on February 1, 1960, with severe abdominal pain. In 1937 he had suffered a perforated peptic ulcer and in 1954 a Billroth I partial gastrectomy had been carried out for continued symptoms. Two days prior to admission he had fallen against a bus seat and injured his left chest. He had complained of a pleuritic pain since then. On the day of admission he had been seized with severe abdominal pain which commenced across the lower abdomen and rapidly became generalized. The pain was colicky and accompanied by vomiting. He had not had a bowel action for 2 days and passed no flatus after the onset of the pain.

ON EXAMINATION.—He was in severe pain and looked very ill with hippocratic facies. He was not shocked; there was no peripheral cyanosis. The pulse-rate was 80 and the blood-pressure was 130/80 mm. Hg. There was generalized abdominal tenderness and rigidity but these signs were more marked on the left side. Bowel sounds were present. Generalized peritonitis was diagnosed.

LAPAROTOMY.—There was a considerable amount of clear fluid with an offensive odour. The colon was found to be dilated and intensely congested. Black and green mottled patches of gangrene were situated over the colon as shown in *Fig.* 198. Some adjacent areas of colon appeared quite normal. The gangrenous bowel had the consistency of wet blotting paper. The colic vessels were pulsating and a small artery on the surface of the colon bled vigorously when deliberately cut to assess the vascularity. The affected bowel was excised through a separate incision and the distal colostomy brought out through the lower end of the paramedian wound.

Examination of the mucosa (*Fig.* 198) showed it to be a greyish-black colour. There was no ulceration. The mucosa of the proximal colostomy showed similar appearances, but no further resection of this was carried out.

HISTOLOGY.—Examination (*Fig.* 199) showed necrosis of the mucosa with loss of staining properties. There was marked œdema of the bowel wall and a diffuse infiltration with polymorphonuclear leucocytes. The vessels were congested but there was no evidence of thrombosis, embolism, or vascular disease. Gram staining showed vast numbers of Gram-positive rods which had colonized almost entirely within the submucosa (*Fig.* 200).

POST-OPERATIVE COURSE.—For the first week following operation he progressed well. Within 48 hours the mucosa of the colostomy recovered considerably and

looked more normal. He was treated during this period with intravenous terramycin and blood transfusion. During the second week a right upper lobe pneumonia developed and *Proteus vulgaris* was cultured from the sputum. This responded to antibiotic treatment. Two weeks after operation he appeared suddenly to develop an enterocolitis and within a few hours he lost almost 5 l. of fluid from the colostomy. Culture of this fluid revealed a mixed growth of *P. vulgaris* and *B. coli*. This infection was controlled with antibiotic therapy and fluid replacement. Following this he developed huge ulcers over pressure areas, despite vigorous treatment, and these have required skin-grafting.

DISCUSSION

Comment on Cases.—These 6 cases appear to be examples of the same pathological process producing a uniform clinical picture. There were 5 females and 1 male and their ages ranged from 56 to 78 years. They suffered from a catastrophic illness which caused the death of 3 of them. Immediately prior to the onset of symptoms they had been well, although *Cases* 2, 4, 5, and 6 complained of varying degrees of constipation.

Clinical Presentation.—Abdominal pain came on suddenly and was constant and very severe. In 3 patients it remained central and in 3 it became generalized having spread from the lower abdomen. The pain was accompanied by bilious vomiting. None of these patients had passed flatus. None had diarrhœa, and none had blood in the rectum. On examination all were readily recognized as surgical emergencies requiring laparotomy. The character of the pain suggested a perforation of an abdominal viscus. Five patients were cyanotic, 4 of them being in a state of collapse with cold extremities and 2 severely hypotensive. Tenderness and rigidity were generalized in 3 cases, more central in 2, and not very marked in 1. Abdominal distension was noted in 5 cases and free fluid demonstrable in 2.

A provisional diagnosis of generalized peritonitis was made in 4 patients, strangulated intestinal obstruction in 1, and intra-abdominal vascular catastrophe in 1.

Findings at Operation.—At laparotomy a few hours after the onset of symptoms all patients had large amounts of putrid-smelling, non-hæmorrhagic fluid within the peritoneal cavity. There was distension of the colon, most marked in *Case* 3, as illustrated by the pre-operative X ray, *Fig.* 187. The distribution of the gangrene seen on the serosal aspect of the bowel is shown in *Fig.* 182. The patches of mottled grey-green necrosis were constant features and represented full-thickness gangrene of the bowel wall. The consistency of the affected areas was that of wet blotting paper and the areas appeared to be scattered haphazardly over the surface of the colon, without relation to the blood-supply or venous drainage. The adjacent bowel was intensely congested and œdematous.

Assessment of the blood-supply and venous drainage was difficult because the mesentery was œdematous and the patients too ill for prolonged examination of the vessels. In *Cases* 1 and 4 it was thought that the pulsations in some mesenteric vessels were absent.

In 4 cases the mucosal surface of the diseased bowel was examined immediately after resection. The appearance is shown in *Fig.* 194. This swollen, gelatinous, black-brown mucosa was in striking contrast to the serosal appearances of the bowel. There was no ulceration of the mucosa and no blood or exudate in the lumen. The mucosal change was continuous, and extended for a considerable distance beyond the necrotic areas seen on the serosal aspect.

Histology.—Microscopy demonstrated identical changes in each case differing only in degree. The bowel showed necrosis almost entirely limited to the mucosa. The architecture of the tubular glands was completely lost and there was a polymorphonuclear cell infiltration which was least marked in *Case* 3 (*Fig.* 189), and most marked in *Case* 2 (*Fig.* 185). These changes affected the submucosa to a lesser extent and here œdema was very noticeable. The blood-vessels were enormously dilated and congested, but there was no evidence of primary thrombosis or vascular disease. An intensive search in the mesentery of the specimens revealed a thrombus in a very small vein in *Case* 2. Autopsy subsequently showed this was isolated and not significant.

Aetiology.—

Infarction.—The dramatic onset of gangrene in these patients suggested the disease had a vascular origin and indeed infarction due to mesenteric vascular occlusion was the diagnosis made at operation in 4 of the 6 cases. Based on the gangrenous changes in the bowel wall and the apparent loss of pulsation in some of the colic vessels, this diagnosis was revised in those 4 cases when no significant arterial or venous occlusion could be demonstrated in the histological material or in 2 autopsies.

Infarction of the large bowel alone is rare, and only 27 cases have been found in the literature: Lauenstein (1882), Faltin (1912), Trotter (1913), Dzialoswynski (1925), Cokkinis (1926), Brown and Dey (1932), Ferguson (1932), Gambee (1937), Moore (1941), Huggard and Herstein (1950), Lushbaugh, Rubin and Rothman (1948), Johnson and Baggenstoss (1949b), Shaw and Green (1953), Forty (1957), Cooling and Protheroe (1958), Ende (1958), Holsey (1958), Rijsbosch (1958), McCort (1960), Blair and Duguid (1960). This is undoubtedly due to the good collateral circulation provided by the marginal vessel conveying blood from the superior mesenteric artery. Ligation of the inferior mesenteric artery is now established in the surgical treatment of aortic aneurysm and carcinoma of the rectum and in such cases impairment of the circulation to the colon rarely occurs if the marginal artery inflow is adequate. Shaw and Green (1953), Harkins (1936), and others have reported cases in which thrombosis of the inferior mesenteric artery has occurred without infarction of the colon.

Occlusion of the inferior mesenteric vein is also less likely to produce infarction of the bowel than occlusion of the superior mesenteric vein. Johnson and Baggenstoss (1949a) examined 99 autopsies in which mesenteric venous thrombosis had occurred. Infarction of the colon followed thrombosis of the inferior mesenteric vein in 16 per cent, whereas infarction of the small bowel occurred in 52 per cent of the cases of superior mesenteric venous thrombosis.

On closer examination there are differences between the clinical presentation of large bowel

infarction and these 6 cases. The sexes appear to be equally affected in large bowel infarction. There are frequently predisposing factors such as ischæmic heart disease, cardiac arrhythmias, portal hypertension, and abdominal surgery. None of these was present in our cases. Patients who suffer a mesenteric thrombosis do not always come to hospital as quickly as did these 6 patients.

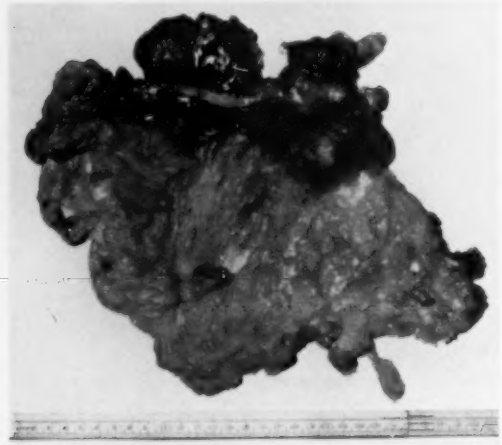

FIG. 201.—Infarction of transverse colon. This shows a specimen of transverse colon and greater omentum. Infarction of the colon had occurred subsequent to arterial obstruction. The colon and adjacent tissue were engorged with blood. This illustrates the contrast between infarction and the necrosis illustrated in *Figs.* 188 and 194. (*By courtesy of Mr. D. B. Craig.*)

Harkins (1936) found that the average duration of symptoms before coming to hospital was eight days in patients suffering from small bowel infarction. McCort (1960) reported 3 cases of large bowel infarction in which the duration of symptoms was 21 days, 3 days, and 2 days. In severe cases of infarction the collapsed state of the patient usually suggests an internal hæmorrhage (Harkins, 1936). There is no cyanosis or toxæmia. Rectal bleeding occurred in 7 of 18 adequately reported cases of large bowel infarction in the literature. It did not occur in any of our 6 patients. Peritoneal irritation is not always marked in mesenteric thrombosis: Jenson and Smith (1956) drew attention to this. In 4 of our cases, however, a pre-operative diagnosis was made of generalized peritonitis.

A review of the literature indicates that the macroscopic appearance of infarction is identical whether it occurs in large or small bowel. A typical hæmorrhagic infarction results. The bowel is red, purple, or black, and is considerably thickened and œdematous. It is engorged with blood (Moore, 1941), and its consistency has been likened to a 'tube of rubber' by Desplas (1927). There is frequently a blood-stained peritoneal exudate and always a considerable loss of blood into the lumen of the bowel. *Fig.* 201 shows these features in a case where infarction of the transverse colon occurred. The hæmorrhagic nature of the infarcted bowel and mesentery is seen. There were 2 pints of almost pure blood in the peritoneal cavity. The gangrenous bowel in our 6 patients, however, was quite different. There was no hæmorrhagic engorgement of the bowel wall which was thin and friable. There was no blood in the lumen of the bowel and the peritoneal exudate was not blood-stained. The necrotic areas were grey-green in colour. *Cases* 2, 3, 5, and 6 exhibited a patchy distribution which is most unlike intestinal infarction and in no case did this distribution correspond to the blood-supply or venous drainage. The extensive mucosal changes were a striking contrast to the serosal aspect of the bowel when examined in *Cases* 2, 4, 5, and 6, and are well illustrated in *Fig.* 194. Hæmorrhagic bowel infarction has a more uniform appearance affecting the mucosal and muscle layers equally.

Evidence of vascular occlusion sufficient to cause the extensive gangrene was not found in these patients. *Case* 1 was not very satisfactory as the description at operation stated "some colic arteries pulsated and some did not". The patient died and autopsy was not performed. In the remaining patients vascular occlusion was adequately looked for but not found. In *Case* 6 a small artery on the surface of the affected colon bled vigorously when it was deliberately cut. A review of the 27 examples of large bowel infarction found in the literature reveals that vascular occlusion was demonstrable in 22 of them, or 81·5 per cent. Jenson and Smith (1956), reviewing small bowel infarction, found vascular occlusion in 41 out of 51, or 80·4 per cent.

Infarction of small bowel has been reported where no vascular occlusion could be demonstrated by surgeon or pathologist (Jenson and Smith, 1956; Glotzer and Shaw, 1959). In some cases, of course, there are technical difficulties. Extensive dissection of the mesentery at operation may not be justified in very ill patients. In surgical specimens the depth of mesentery excised and available for pathological study is often small and in fatal cases autopsy may not be carried out. There remains, however, a group in which adequate examination is carried out and no vascular obstruction is found. Glotzer and Shaw (1959) reported 9 such cases out of 31 autopsies at the Massachusetts Hospital. As these patients had a high incidence of atherosclerotic narrowing of the mesenteric vessels, congestive cardiac failure, and cardiac arrhythmias, it seems likely that a poor cardiac output had precipitated acute ischæmic changes in the bowel wall.

Glotzer and Shaw (1959) compared this possibility to symmetrical peripheral gangrene affecting the extremities which occasionally complicates myocardial infarction. None of our 6 patients appeared to suffer from these predisposing factors, and the examples of infarction without demonstrable vascular occlusion in the literature were all hæmorrhagic in appearance.

The histological appearances of our cases do not in themselves absolutely exclude infarction. A gangrenous process has occurred with most marked changes in the mucosa. The intense polymorphonuclear response as seen in *Case* 2 (*Fig.* 185) suggests that tissue necrosis has resulted from infection rather than ischæmia. The absence of thrombosis or vascular disease in the vessels throughout the bowel wall suggests that gangrene has not followed a primary vascular obstruction or secondary thrombosis associated with Buerger's disease or polyarteritis nodosa.

Lushbaugh, Rubin, and Rothman (1948) described a hæmorrhagic infarction of the colon in a patient who suffered from scleroderma. The histological changes in the bowel wall were characteristic of this disease. The submucosa and muscle were largely replaced by fibrous tissue and endarteritis was widespread.

There are 2 cases in the literature reported as infarction of the large bowel where the usual hæmorrhagic changes have not occurred and the description of the macroscopic appearances resembles our cases. Thompson in 1948 described a case of acute gangrene affecting the sigmoid colon in a male aged 56 years. Malodorous murky fluid was found in the peritoneal cavity and greyish-black patches were noted over the sigmoid colon without any effusion or blood into the bowel. He described this as an 'ischæmic' rather than hæmorrhagic infarction. The histology showed patchy necrosis affecting the bowel wall which in some areas affected the mucosa only. There was an acute inflammatory reaction around these areas. Veins in the muscle layers and on the surface of the bowel were thrombosed. There was no thrombosis in the main mesenteric vessels and Thompson suggested that this infarction was due to a primary intramural vein thrombosis. Russell (1950) reported a case of 'anæmic infarction' of the descending colon in a diabetic female of 73 years. The bowel in this case was flaccid and slate-grey in colour; there was no exudation of blood into the bowel wall, lumen, or peritoneal cavity. Russell suggests the cause was narrowing of the inferior mesenteric artery due to atherosclerosis. A fine probe, however, could be passed through the narrowed segment of the artery.

We suggest that these cases and others may have been interpreted as examples of infarction where the evidence for this diagnosis is not complete. The clinical and morphological features seem to bear a close resemblance to the 6 cases we have described, and in these 6 patients the evidence is strongly against the diagnosis of infarction.

Ulcerative Colitis.—These cases do not appear to be a variety of ulcerative colitis. The fulminating forms of ulcerative colitis produce a very acute illness always accompanied by a purulent bloody diarrhœa. The changes in the bowel wall represent inflammation and not necrosis. Ulceration of the inflamed mucosa is marked and a purulent exudate is present. The histological appearances in our cases bear no resemblance to the changes seen in ulcerative colitis (Drury, 1960; Morson, 1960).

Staphylococcal Enterocolitis.—This produces a profound toxæmia accompanied by abdominal distension. Diarrhœa nearly always occurs although it can be delayed. Most of these cases have received antibiotics and huge numbers of staphylococci are present in the stools (Webster, 1958). The small bowel is grossly inflamed as well as the colon.

Pseudomembranous Enterocolitis.—This disease affects the small bowel as well as the colon. There is a necrotizing inflammation affecting mainly the mucosa (Corridan and Shucksmith, 1958) and all cases show the characteristic formation of a yellowish-white friable pseudomembrane which is composed of necrotic cellular debris, fibrin, pus cells and clusters of Gram-positive cocci and Gram-positive rods. There is ulceration of the mucosa. Diarrhœa almost always occurs.

Infection.—Infection of the bowel was considered, as at operation the serosa was intensely inflamed and associated with considerable œdema in the mesentery. The histology revealed an intense polymorphonuclear infiltration of the submucosa and so Gram stains of the tissues were made. These showed invasion of the mucosa and submucosa in all specimens with an enormous uniform colony of Gram-positive rods 3–4 μ in length. In *Fig.* 193 the organisms are seen within submucosal veins and in *Figs.* 197 and 200 they are exclusively within the submucosa.

On morphological evidence alone these are certainly *Clostridia* and probably *Cl. welchii*.

Two points suggest these organisms are not merely secondary invaders of necrotic bowel. Firstly, there is an intense polymorphonuclear response in the submucosa which would indicate a reaction of living tissue to an infecting organism, and, secondly, the organisms are seen in greatest numbers in the submucosa and deeper parts of the mucosa. The complete absence of bacteria in the necrotic mucosa seen in *Figs.* 197 and 200 strengthens the view that this is not secondary invasion from the bowel lumen. It is emphasized that the 6 surgical specimens were fixed in formalin immediately following resection.

Cl. welchii is widely distributed in nature and is a common inhabitant of the alimentary tract of man. Dudgeon (1926) cultured it from human fæces in 35 per cent of subjects examined. Williams (1926) demonstrated in man and experimental animals that *Cl. welchii* multiplied rapidly in small bowel proximal to an obstruction. Wound gas gangrene infections can rarely follow a number of operations on the alimentary tract, particularly a colostomy, drainage of a paracolic abscess, and appendicectomy. One of us (K. L. W.) has seen gas gangrene of the abdominal wall following resection for a low small bowel obstruction.

In animals, clostridial infections of the alimentary tract such as braxy, lamb dysentery, and enterotoxæmia have been known for a long time and are invariably accompanied by a toxæmia which is rapidly fatal without treatment (Stableforth and Galloway, 1959).

These clostridial infections in man and animals are noted for the profound toxæmia which they produce. The toxæmia our six patients suffered was very severe and was consistent with an overwhelming bacterial infection. Further, they appear to be closely related to an endemic disease of North Germany which was described as 'necrotic jejunitis' by Fick and Wolken (1949) and 'enteritis necroticans' by Zeissler and Rassfeld-Sternberg (1949). The former authors reported 18 cases in which the disease had affected varying lengths of jejunum and had in some cases spread proximally to involve the duodenum and distally to the terminal ileum and adjacent colon. The patients presented with a very acute illness in which sudden severe abdominal pain and vomiting were accompanied by a profound toxæmia and peripheral circulatory collapse. As the disease progressed, diarrhœa, sometimes blood-stained, supervened. Most of the patients had clinical evidence of

peritonitis and came to laparotomy. The affected bowel wall was very congested and turgid with œdema, giving it a 'sausage' appearance. The mucosa appeared 'cauterized' and in others it had sloughed, producing ulceration. Histological examination showed extensive necrosis of the mucosa, which extended into the submucosa in some areas. Œdema and polymorphonuclear infiltration were also marked features. Gram staining of the sections showed many organisms in the tissue, but these were not identified and appeared to be a mixed colony. Sixteen of their patients died, a mortality of 88·8 per cent. They suggested that some dietary lack had affected the resistance of the mucous membrane, allowing invasion by organisms not pathogenic under normal circumstances, and this had produced the necrotic change in the mucous membrane.

Siegmund (1948) carefully examined the mucosa in similar cases and agreed that they contained large numbers of bacteria. However, these appeared to be exclusively Gram-positive bacilli.

Zeissler and Rassfeld-Sternberg (1949) examined 3 operation specimens and 8 autopsy specimens from 11 cases of 'enteritis necroticans'. They were particularly interested in the bacteriological aspects. From all these specimens they obtained Gram-positive bacilli closely resembling *Cl. welchii*. They differentiated it on the morphology, appearance of surface colonies, and the remarkable heat resistance of its spores. They proposed the name '*Cl. welchii* type "F"'. The toxins were examined by Oakley (1949) who demonstrated that this organism produced large quantities of β toxin. Zeissler and Rassfeld-Sternberg (1949) reproduced the disease by introducing 5 ml. cultures into the intestine of guinea-pigs.

Fethers (1959) has recently reported a further case of jejunal necrosis which occurred in a male aged 76 years who was admitted to hospital with a bleeding duodenal ulcer. This case bears an even closer resemblance to our 6 cases than those already discussed. This patient had been admitted with a small hæmatemesis and melæna. The following day he complained of upper abdominal pain and on examination there was tenderness and guarding in the upper abdomen. It was noted that the patient looked more ill than his blood-loss would have suggested. Laparotomy was carried out and on opening the peritoneum a foul odour was noted. A 12 in. loop of jejunum was found to be gangrenous with a greenish-black discoloration on its surface. The mesenteric vessels were pulsating. A chronic duodenal ulcer was present. The gangrenous bowel was excised and an end-to-end anastomosis carried out. Following operation, the patient rapidly deteriorated and died five hours later. The resected specimen was opened and the mucosal gangrene was seen to extend further than was apparent from the external appearance. Histological examination of the resected jejunum showed an acute inflammatory necrosis of the mucosa. The vessels in the submucosa were dilated. Gram staining revealed large numbers of Gram-positive bacilli in the necrotic mucosa. A culture of the bowel produced a growth of *Cl. welchii*.

It is worthy of note that this patient did not have diarrhœa and there was no ulceration of the mucosa. This case appears to link the pathology of the patients described by Fick and Wolken (1949), Zeissler and Rassfeld-Sternberg (1949), and our 6 cases where the necrotic process affected the colon. The absence of ulceration and diarrhœa in these 6 cases and the case Fethers (1959) has described could be explained by a more fulminating infection which would bring patients to laparotomy before the dead mucosa has sloughed.

We suggest that the evidence strongly favours an infective cause for these cases of necrosis of the colon, even though we have not isolated the organism in culture.

TREATMENT

This cannot be on a scientific basis until the cause of the disease is proven. However, on examining the course of this disease and its response to various methods of treatment, certain points emerge.

These cases are readily recognized as abdominal emergencies requiring laparotomy and this should be carried out as soon as possible. Pre-operative resuscitation with blood transfusions may be necessary. Three patients were in a state of shock prior to operation, 2 of them having a profound fall in blood-pressure. *Case* 2, whose pre-operative blood-pressure was 85/65 mm. Hg, was given an infusion of saline only prior to surgery, and subsequently died in a collapsed state as the operation was completed.

There is little doubt that resection of the gangrenous bowel should be carried out as speedily as possible as these were all severely ill patients. *Case* 3 was seen to improve markedly when the dead bowel had been excised and this seems to indicate that the toxæmic effects of the necrotic tissue must have been considerable. The resection should be beyond all gangrenous tissue, particular care being taken not to overlook any small areas of full-thickness gangrene situated some distance from more obvious gangrenous changes. *Fig.* 188 shows these outlying areas on the transverse colon in *Case* 3. Another consideration in favour of wide resection is the disproportion in the extent of the necrosis affecting the mucosa and that seen on the surface of the bowel. Macroscopically, and histologically the mucosal changes extend beyond those in the muscle. In *Case* 5, when the bowel clamp was removed prior to making a right transverse colostomy, the mucosa appeared black and necrotic. Further bowel was prolapsed until normal mucosa was obtained and a further resection then enabled the colostomy to be made with viable bowel. Progressive gangrene occurred following operation in *Case* 1 when the edges of the colostomy became necrotic within 48 hours. It is difficult to estimate how often this is likely to occur as the examples are few, but it does appear to emphasize the need for generous excision of the diseased bowel.

We suggest that construction of a proximal and distal colostomy is preferable to anastomosing the divided ends of bowel. This greatly reduces the operating time and would seem to be safer. *Cases* 3 and 4 were resected with end-to-end anastomosis. Both these patients developed anastomotic leaks and this was the cause of death in *Case* 4.

Because of the possible infective nature of the disease the systemic use of a broad spectrum antibiotic is recommended as a routine following

operation. It seems reasonable to combine this with polyvalent gas gangrene antitoxin in large doses intravenously.

CONCLUSION

We have described 6 cases of a fulminating disease of the colon presenting with abdominal pain, peritonitis, and toxæmia, which we believe is caused by a type of *Cl. welchii*. At laparotomy a characteristic picture is seen with malodorous non-hæmorrhagic fluid associated with a patchy green gangrene of the colon. There is a striking contrast between this serosal aspect of the bowel and the black gangrenous appearance of the mucosa which is more extensive.

This disease would seem to belong to the same group as the 'necrotic jejunitis' described by Fick and Wolken (1949). As the changes in the bowel consist of extensive necrosis accompanied by an inflammatory reaction, the name 'necrotizing colitis' is proposed.

We are fully aware of the limitations of a bacteriological diagnosis without isolating the organism in culture. Until this is done the cause of the disease will remain unproven. It is suggested that in a further case immediate anaerobic culture of the contents of the lumen and the bowel wall should be carried out. Assaying for clostridial toxins from the unfixed specimen is a further method of investigation which should be considered.

SUMMARY

1. Six cases of acute gangrene of the large bowel seen between August, 1957, and February, 1960, are presented.

2. The evidence presented suggests that these 6 cases are of an identical disease process, hitherto not reported.

3. The cause is probably a *Cl. welchii* infection, but this has not been proven by culture or toxin assay.

4. The name 'necrotizing colitis' is suggested as the disease process is characterized by necrosis with an intense inflammatory response.

We would like to thank Mr. T. G. I. James, Mr. F. A. Henley, Mr. J. D. Fergusson, Mr. L. Lockhart Mummery, and Mr. G. Westbury, who kindly allowed us to report their patients. We are grateful to Dr. R. Drury, Dr. B. Morson, and Dr. Drew Thompson, who have reviewed the histological material. Dr. J. D. Allan Gray has examined all the Gram stains and given us advice on the bacteriological problems.

We are indebted to Mr. T. G. I. James, Professor Ian Aird, and Dr. F. Avery Jones for their advice, encouragement, and criticism. Mr. F. Forty and Mr. D. B. Craig kindly allowed us access to the records and the use of the photograph of the case shown in *Fig.* 201.

We are especially grateful to Miss M. Barber for the paintings and diagrams and to Mr. A. Booker for the photographs.

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HERNIATION THROUGH MESENTERIC AND MESOCOLIC DEFECTS

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VERY occasionally intestinal obstruction is due to herniation of a loop of bowel through a naturally occurring defect in the mesentery of the small intestine, but no record has been found in the literature of obstruction caused by the passage of a loop of small intestine through a defect in the sigmoid mesocolon. The following report is of such a case.

CASE REPORT

Mr. C. F., a 35-year-old bank messenger, was admitted to St. Bartholomew's Hospital on March 9, 1956, with several hours' history of central abdominal colic steadily becoming more severe. There was no history of previous operations or attacks of abdominal pain and his general health was good. After admission he began to vomit bile-stained fluid and was rolling about with the pain. The abdomen was soft, with slight tenderness but no rebound sign. There was a faint succussion splash and obstructive bowel sounds. Hernial orifices were normal. There was pelvic tenderness on rectal examination.

AT OPERATION.—A loop of ileum was found to be obstructed as it passed through a hole in the sigmoid mesocolon (*Fig.* 202). The loop was congested but viable and was reduced without great difficulty. It was not adherent to the margins of the defect which were rounded, slightly thickened, and opaque. The aperture was circular, about 1 in. in diameter, and somewhat nearer to the bowel than to the root of the mesocolon. The colon and the rest of its mesentery were normal in appearance. The defect was obliterated by suture.

Post-operative recovery and healing were uneventful.

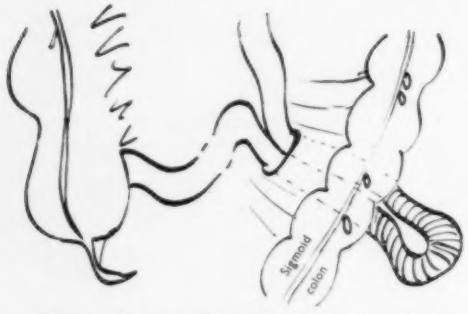

FIG. 202.—Diagram of the operative findings in the case reported. A loop of small bowel has herniated through a defect in the sigmoid mesocolon.

DISCUSSION

Aetiology.—Treves (1885) in an account of defects in the mesentery of the small intestine considered that a minority, due to violence to the abdomen, were slit-like, irregular, and of uncertain situation; but most of the holes he studied were round, situated in the mesentery of the most terminal ileum, and had distinct margins, often thickened and opaque. He suggested that these apertures were either congenital or were acquired by atrophy or rarefaction in a particular congenitally weak area in the mesentery near the bowel in the ileo-cæcal region. The mesenteric rent in the case reported by Brown (1920) was almost certainly due to trauma, but in only 7 of the 28 cases collected by him and by Cutler (1925) was there any suggestion of injury in the history. It seems to be fairly generally agreed that most of these defects are congenital, and cases of obstruction in the neonatal period have been reported by Rutherford (1909) and by Long (1927), in whose patient there were multiple fenestrations in the ileal mesentery.

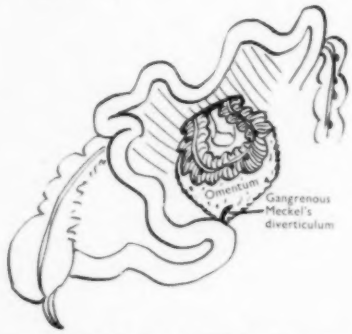

FIG. 203.—Hamaker (1914). Prolapse of transverse colon through a defect in the small bowel mesentery. The omentum is wrapped round a gangrenous Meckel's diverticulum.

King (1935) described a case of recurring appendicitis in which the apex of a loop of small bowel had apparently been drawn through the mesentery by its adhesion to the tip of the appendix. He suggested that inflammatory episodes, usually in the appendix, might account for many of the mesenteric defects and herniæ. Dolton (1944) doubted that inflammation was a significant factor, but reports which might be thought to support King's hypothesis are those of Hamaker (1914), Atherton (1897), and Pye-Smith (1889), all involving Meckel's diverticulum. In Hamaker's (1914) case transverse colon and omentum were found to have prolapsed through a defect in the small bowel mesentery, the omentum enveloping a gangrenous Meckel's diverticulum (*Fig.* 203). In the case reported by Atherton (1897) the loop of ileum which had herniated through a mesenteric aperture bore a twisted and partly gangrenous Meckel's diverticulum which was adherent to the anterior abdominal wall (*Fig.* 204). Pye-Smith (1889) described a case in which a Meckel's diverticulum passed through a hole in the mesentery of its own portion of ileum, encircled the bowel closely and was firmly attached by its tip to the mesentery above the hole (*Fig.* 205).

Protagonists of the congenital theory of these mesenteric defects might argue, however, that these 3 cases merely illustrate that congenital abnormalities are frequently multiple. On the other hand, peptic

ulceration is found in a considerable proportion of cases of herniation of small intestine through the transverse mesocolon and the associated inflammation and adhesions may well be of aetiological significance in at least some of the cases (Heaney and Simpson, 1925; Tanner, 1945). Herniation through defects resulting from operations such as gastro-enterostomy is, of course, excluded from consideration. Hogarth Pringle (1919), on the evidence of a variety of cases, suggested that the area of transverse mesocolon to the left of the middle colic vessels may be, like Treves's 'field' in the terminal mesentery, a site of congenital thinning and consequent susceptibility to aperture formation.

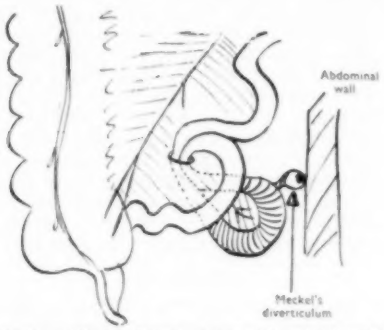

FIG. 204.—Atherton (1897). A loop of terminal ileum has herniated through a mesenteric defect. The Meckel's diverticulum which is on the loop is twisted, partly gangrenous, and adherent to the anterior abdominal wall.

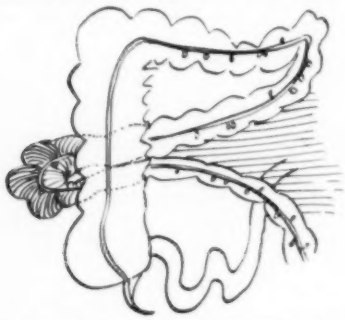

FIG. 206.—Cullen (1936). The descending colon has passed through the ascending mesocolon.

The mesosigmoid defects in the case now reported and that of Turel (1932) had smooth, rounded, thickened edges, and must be considered of long standing and possibly congenital. This was Turel's opinion of his own case although the defect was a slit extending from the bowel to the base of the mesosigmoid. Parson (1899) reported a case of strangulation of small bowel through the lower of two oval windows in an abnormal fold of peritoneum on the left side of the mesosigmoid. The fold, which was a double layer and contained branches of sigmoid vessels, was undoubtedly a congenital abnormality.

Site of Defect.—Most of the defects reported have been in the terminal mesentery in Treves's 'field', but involvement of more proximal mesentery has been recorded several times. The ascending colon is not infrequently mobile and suspended on a shorter or longer mesentery, and herniation of bowel through both leaves of this ascending mesocolon has been described by Cullen (1936) (*Fig.* 206) and by Hansmann and Morton (1939) (*Fig.* 207). The transverse mesocolon is the commonest site of mesocolic defects (Menegaux, 1934), and Heaney and Simpson (1925) summarized 21 cases of herniation of

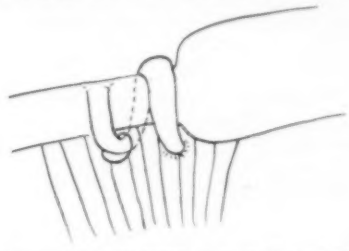

FIG. 205.—Pye-Smith (1889). A Meckel's diverticulum has passed through a defect in the mesentery and after closely encircling the bowel has become attached by its tip to the mesentery above the defect.

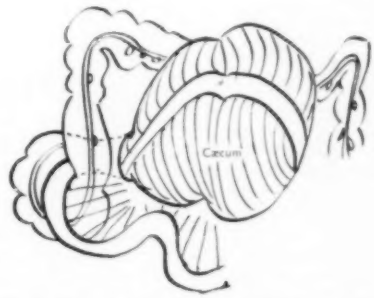

FIG. 207—Hansmann and Morton (1939). The cæcum is obstructed where it has passed through the medial leaf of the ascending mesocolon.

FIG. 208.—Turel (1932). Obstruction due to prolapse of a loop of sigmoid colon through a defect in its own mesocolon.

small bowel through this site. In one of these the intestine re-emerged through the foramen of Winslow; in 8 cases the bowel re-entered the general peritoneal cavity by rupture through the gastro-hepatic omentum, and in 3 it traversed the gastro-colic omentum; in the remaining cases the hernia was

confined to the lesser sac. Defects in the transverse mesocolon are usually on the left side, but in Heaney and Simpson's (1925) first personal case and in that of Smith (1945) the aperture was on the right. The descending colon is rarely sufficiently mobile to allow herniation behind it, but the sigmoid mesocolon is not quite immune from this type of internal hernia (Turel, 1932) (*Fig.* 208).

Site of Obstruction.—The obstruction is usually in the small bowel and may be due to herniation through mesentery of small intestine, through

FIG. 209.—Judd (1929). Herniation of right colon and terminal ileum through a defect in the small bowel mesentery in a case of failure of normal intestinal rotation.

transverse mesocolon, or, as in the case now reported, through the mesosigmoid. The right colon was involved in the hernia in two reports. In Judd's (1929) case the ascending colon, cæcum, and terminal ileum were obstructed by herniation through an aperture in the small bowel mesentery (*Fig.* 209). It is obvious from the position of the parts in this case that there had been a failure of normal rotation of the intestine during development. Hansmann and Morton (1939) reported a case in which the cæcum was obstructed and enormously dilated by herniation through the ascending mesocolon from lateral to medial side (*Fig.* 207). There was post-mortem evidence of a large long-standing retroperitoneal hernial sac which had contained terminal ileum and cæcum, and from which the latter had escaped by rupture of the medial leaf of the ascending mesocolon. The transverse colon was involved in the transmesenteric hernia reported by Hamaker (1914) (*Fig.* 203), and the descending colon was found to have passed behind the ascending colon in the case described by Cullen (1936) (*Fig.* 206). Turel (1932) recorded a case in which the sigmoid colon was obstructed as a result of herniation through a defect in its own mesentery (*Fig.* 208).

A viscus other than a loop of bowel may pass through a mesenteric defect and cause intestinal obstruction. In Pye-Smith's (1889) case a Meckel's diverticulum closely encircled the bowel (*Fig.* 205), and McWhorter (1928) reported a case in which the greater omentum had passed through a defect in the root of the small bowel mesentery and had become adherent to itself to form a ring.

Clinical Features.—There are no special features which distinguish these cases from any other form of internal hernia associated with obstruction. Several of the cases reported were in boys before or about the age of puberty. Neonatal cases were recorded by Rutherford (1909) and by Long (1927). Franklin's (1894) patient was 73 years old.

SUMMARY

A report is presented of a unique case of intestinal obstruction due to herniation of a loop of small bowel through a naturally occurring defect in the sigmoid mesocolon. The aetiology and location of mesenteric and mesocolic defects are discussed and the types of obstruction which have been reported are reviewed.

Acknowledgement.—I wish to thank Mr. Rupert Corbett for permission to report this case, and for his helpful criticism and advice.

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CONTINUOUS RECORDS OF GASTRIC *p*H IN SITU AND THEIR POSSIBLE USE IN PRE-OPERATIVE ASSESSMENT OF PEPTIC ULCER PATIENTS

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THE gastric contents have been examined in many different ways but little information is available about continuous records of gastric *p*H using an electrode in the stomach. This paper recounts experiences with such a method and their possible application to the pre-operative assessment of peptic ulcer patients.

HISTORICAL

Some 45 years ago McClendon (1915a, b, c) developed a hydrogen electrode which could be swallowed and he found that gastric *p*H varied before, during, and after ingestion of food. Little notice was taken of this work until 1939–41 when a few authors (Eyerly and Breuhaus, 1939; Flexner and Kniazuk, 1939; Eyerly, 1940; and Flexner and Kniazuk, 1940, 1941) used a glass electrode in the stomach of the dog.

As a possible alternative to glass, Haggard and Greenberg (1941) tried an antimony electrode. Such

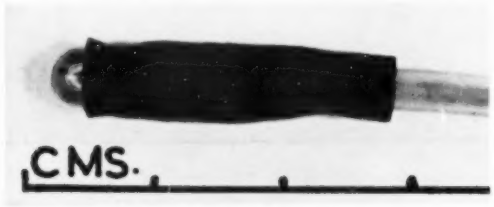

FIG. 210.—Stomach glass electrode.

electrodes were also used with varying success by German workers (Kreitner, 1949; Kreitner and Pantlitschko, 1949a, b; Pantlistchko and Schmid, 1949; Kreitner, 1950; Kinzlmeier, 1951; Kinzlmeier, Henning, and Demling, 1951; Henning, Demling, and Kinzlmeier, 1951; Kinzlmeier, Demling, and Mannuss, 1952; Henning, Kinzlmeier, and Demling, 1952; Demling, Kinzlmeier, and Henning, 1952; Ströder and Kubin, 1954; Kinzlmeier, Henning, and Demling, 1954; Kimbel and Kinzlmeier, 1954; Kimbel, Kinzlmeier, and Bosch, 1954; Henning, Kinzlmeier, and Kimbel, 1954; and Wolf and Kinzlmeier, 1957). The period of examination varied from 2 to 9 hours and different forms of reference electrode liquid junction were used. In one or two patients two electrodes, one in the juice and the other against the wall of the stomach, showed a definite but fairly constant difference between the *p*H in situ and in aspirated samples from the same patient.

In a critical review of *p*H electrodes and equipment, Schwabe (1953) discussed the merits of glass and antimony electrodes, and concluded that antimony was unsuitable for any but brief records because of its tendency to oxidize.

Meantime, the glass electrode had been found suitable for short periods—1–4 hours—by Bradley and Tilghman (1952), Rovelstad, Owen, and Magath (1952), and Rovelstad (1956). Only Schmidt-Kessen (1954a, b; 1955a, b) and Grieve (1955) continued the investigation for 24 hours or longer.

METHODS AND MATERIAL

For *p*H measurements a Cambridge valve potentiometer driving a model B recorder was found suitable. A special gastro-intestinal-type glass electrode was used (*Fig.* 210), and a standard reference

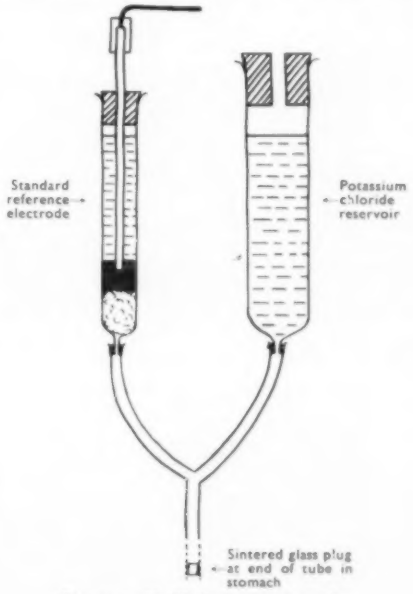

FIG. 211.—Modified reference electrode.

electrode was modified by the addition of a potassium chloride reservoir and a long polyvinyl chloride tube to allow the liquid junction to be in the stomach (*Fig.* 211). The distilled water used for preparing the saturated potassium chloride solution was boiled to eliminate air-bubbles which had proved troublesome during the earlier part of the work. In addition the porous glass plugs used for the liquid junction were cleaned regularly by immersion in glass-cleaning fluid and prolonged washing in distilled water. The equipment was standardized before and after use with buffer solutions of *p*H 1 and *p*H 5 (Gold, 1956).

This apparatus was used for two types of investigation: (1) *in vitro*; (2) *in vivo*.

1. In Vitro.—In this, poisoning, polarization, contamination, and mucosal burial of the glass electrode were examined. To test for poisoning,

glass and reference electrodes were recorded for 3 weeks while immersed in milk and, for polarization, for a similar period while in a standard buffer. Contamination by mucus and burial in mucosal folds were investigated in a post-mortem stomach containing buffer solution of *p*H 1 (*Fig.* 212).

2. In Vivo.—The investigations under this head were in two parts.

a. The Examination in situ of the pH of the Stomach Contents of 51 Patients.—In each patient the glass electrode tube was passed through one nostril and the

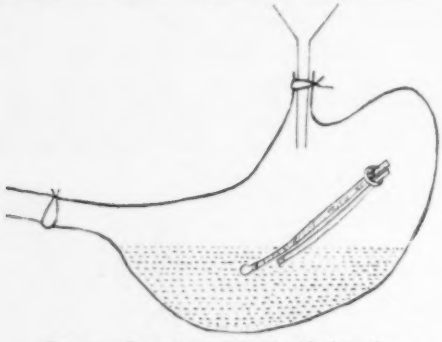

FIG. 212.—Post-mortem stomach with electrodes.

reference electrode tube through the other. The two electrode tubes were then 'fished' from the pharynx with forceps, lashed together, and swallowed by the patient. Great care was necessary to prevent damage to the glass electrode by the patient's teeth or by the forceps used for 'fishing'. The *p*H of the gut content gave an index of the level of the electrodes, whose position was checked by a radiograph of the upper abdomen.

Table I.—DISEASES FROM WHICH PATIENTS SUFFERED

Disease	*Number*
Normal	6
Duodenal ulcer	27
Duodenal + gastric ulcer	2
Duodenal ulcer + gall-bladder	1
Gastric ulcer	4
Gastric cancer	9
Anæmia	1
Gall-bladder	1
TOTAL	51

During the recording each patient was in bed and had ordinary ward diet. The time at which he or she ate, drank, or smoked was noted. In addition, the effect of histamine in standard dosage (0·01 mg./kg. body-weight) and in augmented dosage (4 times standard) with antihistamine cover (Kay, 1953) was studied in some of them.

A 3–4-hour record may be unrepresentative and wherever possible records continued for 24 hours and in a few patients for 48 hours.

b. The Effect of Burial of the Electrodes in Mucosal Folds and a Comparison of Aspirated Samples with the Continuous Record.—In 3 patients undergoing upper abdominal operations electrodes were passed preoperatively and during the operations the stomach was handled and the electrodes buried temporarily in mucosal folds. One patient of the main group swallowed a Ryle's tube lashed to the electrode assembly, and samples were aspirated hourly for comparison with the recorded *p*H from the same site.

RESULTS

1. In Vitro.—During the 3 weeks' run in milk and in standard buffer no change occurred in the records which suggested either poisoning or polarization of the electrodes.

In the post-mortem stomach contamination by mucus did not interfere with the record, and severe friction on the electrode had only a slight temporary effect on readings, and burial in mucosal folds none. The experiment was repeated 3 times with the same result.

2. In Vivo.—

a. The diseases from which the patients suffered are shown in *Table I.* Those in the normal group had no recognizable disease of the alimentary tract; the duodenal and gastric ulcers and gall-bladder disease were confirmed radiologically and often at subsequent operation. Carcinoma was confirmed by histology.

Table II.—MEAN READINGS AND STANDARD DEVIATIONS

Disease	*Mean pH*	*Number of Observations*	*Standard Deviation*
Normal	3·03	6	1·16
Duodenal ulcer	3·16	27	1·73
Duodenal ulcer + gastric ulcer	3·55	2	1·35
Duodenal ulcer + gall-bladder	5·1	1	—
Gastric ulcer	2·74	4	1·42
Gastric cancer	5·86	9	2·09
Anæmia	4·3	1	—
Gall-bladder	6·0	1	—

Figs. 213–216 give examples of the tracings obtained from a normal patient, and from patients with duodenal ulcer, gastric ulcer, and carcinoma respectively. The vertical scale reads 0–10 *p*H in steps of 0·2 *p*H units while the horizontal gives time in hours.

Table III.—RESULTS OF AUGMENTED HISTAMINE TEST

DISEASE	NUMBER	RESPONSE				
		Nil	Transient	Marked/ Quick	Marked/ Slow	Small/ Slow
Normal	3	—	3	—	—	—
Carcinoma	2	2	—	—	—	—
Duodenal ulcer	14	—	2*	2	2	8

* 1 died 2 weeks later.
1 had biliary disease and small duodenal ulcer.

Study of all the patients' records suggests that each patient has a *p*H base-line—a *p*H level to which his or her record returns after any temporary fluctuations. While this appears to be characteristic for the individual it is not diagnostic of a particular disease.

In *Table II* the main readings and standard deviations of the *p*H for the different groups of patients are summarized. In the normal patients and those with gastric and duodenal ulcers there is no great difference between the mean *p*H levels but there is considerable scatter in the results and in general

those with duodenal ulcers show the lowest *p*H base-line, the normals, and those with gastric ulcers a rather higher base-line, and carcinomic patients the highest of all.

Nineteen patients were examined by the augmented histamine test and the results are summarized in *Table III*. Five types of response have been recognized (*Fig.* 217):—

i. Nil.

ii. Transient: little change noted in *p*H.

iii. Marked quick: decrease in *p*H of 0·4 units or more with a quick return to previous levels.

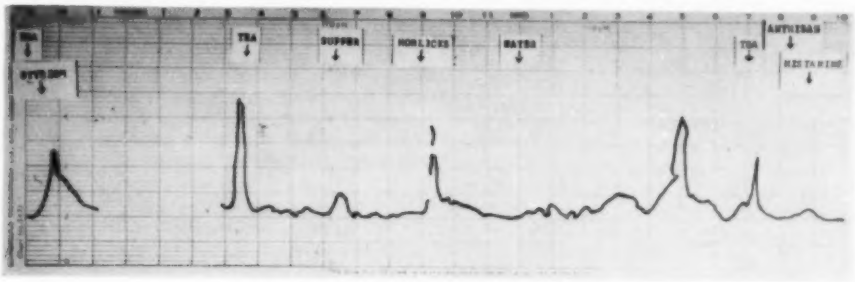

FIG. 213.—Chart of patient without alimentary tract disease.

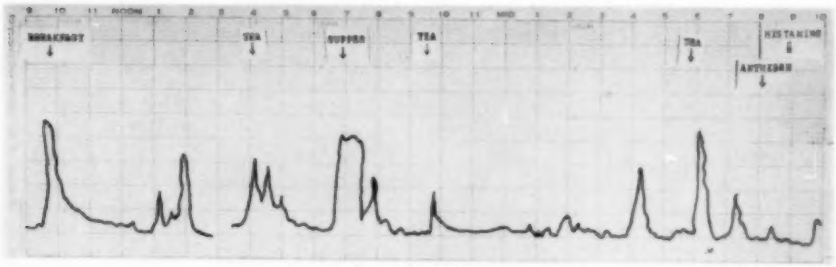

FIG. 214.—Chart of patient with duodenal ulcer.

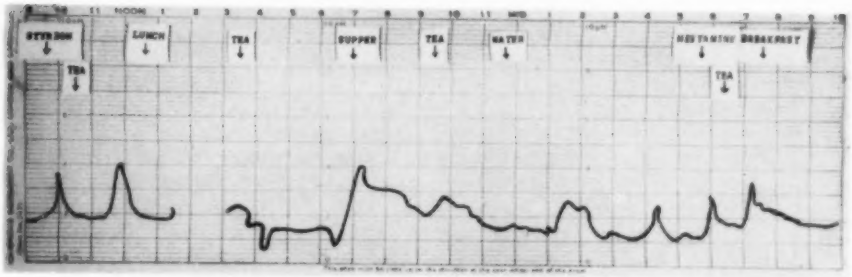

FIG. 215.—Chart of patient with gastric ulcer.

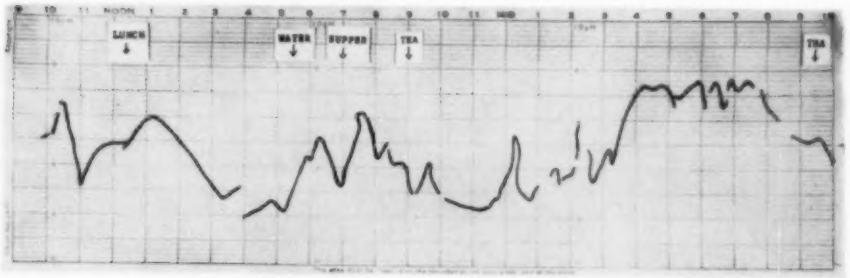

FIG. 216.—Chart of patient with gastric carcinoma.

iv. Marked/slow: decrease in *p*H of 0·4 units or more with a slow return to previous levels.

v. Small/slow: smaller responses, 0·3 *p*H unit or less, with a slow return to previous levels. Examples of (iii), (iv), and (v) appear in *Figs.* 218–220.

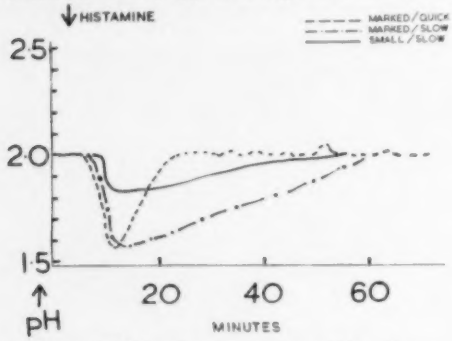

FIG. 217.—Differing responses to augmented histamine test.

All the duodenal ulcer patients who had this test were treated surgically. Those in categories (iv) and (v) showed a good result after operation while those in category (iii), though fairly satisfactory, were not as good as those in (iv) or (v). The type of operation varied (Polya, Billroth I, and vagotomy and gastroenterostomy) and did not appear to have any influence on the result.

b. The effect of handling and burial in mucosal folds in 3 patients at operation is summarized in *Table IV*. The maximum change of 0·5 *p*H unit was caused by extreme friction. When the *p*H of aspirated contents was compared with the in situ *p*H (*Table V*) the aspirated samples were always more acid by between 0·8 and 1·4 *p*H units.

DISCUSSION

The results obtained in these investigations show that the continuous measurement of gastric *p*H in situ in the human subject is possible and that for each individual there appears to be a *p*H base-line. Various authors have suggested ways in which

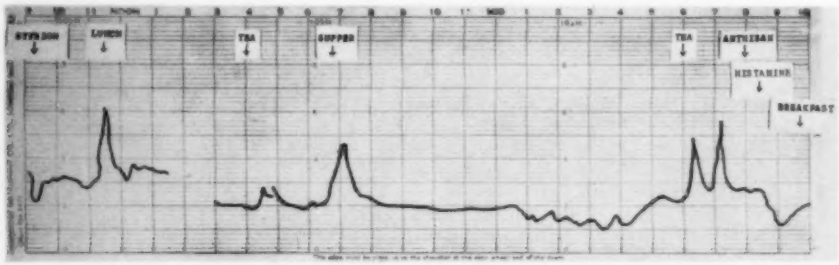

FIG. 218.—Marked response to augmented histamine with quick return to previous level.

FIG. 219.—Marked response to augmented histamine with slow return to previous level.

FIG. 220.—Small response to augmented histamine with slow return to previous level.

failure might occur—polarization (Copp, 1958), poisoning (Cambridge Instrument Company, 1956), contamination by mucus (James, 1957), or burial of the electrodes in mucosal folds (Rovelstad, 1956). No discrepancies from any of these causes were discovered experimentally or in the living patient. The chief trouble to occur was electrical—fracture of the glass electrode lead or failure of one or the other of the potentiometer valves. When interpreting results, it is important to remember that the *p*H scale is logarithmic and that in the range found in the stomach a small change in *p*H represents quite a big change in acidity.

Table IV.—EFFECT IN 3 PATIENTS OF PLACING OF ELECTRODE

*p*H with Electrode Free	*p*H with Electrode Enfolded
4·3	4·3–4·7
7·2	7·3
4·0	4·2

Table V.—COMPARISON OF ASPIRATED SAMPLE AND RECORD *p*H

Time	Aspirated Sample *p*H	Record *p*H
6 p.m.	2·4	3·6
7 p.m.	4·6	5·4
8 p.m.	2·4	3·8
9 p.m.	2·8	3·8
10 p.m.	2·2	3·4

Samples aspirated from the stomach were more acid by 0·8–1·4 *p*H unit than the recorded *p*H at the time of aspiration. This accords with the finding of Flexner and Rossett (1943), Von Hofstetter (1947), Rovelstad and others (1952), and Rovelstad (1956).

Examination of the tracings shows that the *p*H level may be changed temporarily by different substances such as food, water, and alkali (*Figs.* 213–216). The change is of short duration unless food or alkali is given continuously and in large amount, and no satisfactory method of assessment has been found.

In addition, while most of the changes occur immediately after the administration of a substance, in *Fig.* 213 at 5 a.m. and in *Fig.* 214 at 4.15 a.m. there are brief changes for which no real explanation can be offered. The behaviour of the trace is even more odd in *Fig.* 216, where from 3–9 a.m. the *p*H remains at a high level. This patient had carcinoma of the stomach and a rather erratic acid secretion and this may be the reason.

Great emphasis has often been placed on 'acid-rebound' after the administration of large doses of alkali, but on no occasion was this observed in the present group of patients. Alkali caused a temporary rise of *p*H, but when the alkali was stopped the *p*H dropped to its old level but not below it. This point is illustrated in *Figs.* 213, 215 after the administration of the ion-exchange resin polyaminostyrene.

In the 12 duodenal ulcer patients examined by the modified augmented histamine test, 2 showed a marked response to the injection with a rapid return to the *p*H base-line and these gave a less satisfactory result from operation. In the other 10 the return to the *p*H base-line was much slower and the result of operation was better. The result of operation was assessed in terms of presence or absence of 'dumping' discomfort after food, ability to take a normal meal, and fitness for return to previous occupation. All 12 patients showed improvement, but the 2 already mentioned showed moderate dumping and intermittent discomfort after food. A possible explanation for this is that in the small group of 2 there was still good buffering of the stomach contents and operation was therefore less likely to give so much improvement. In the other 10 with the slow return to the *p*H base-line the buffering mechanism was inadequate, and operation helped that group.

In Kay's original test the total acid output of which the stomach is capable is measured, whereas with the present modification an objective assessment is made of the stomach's ability to deal with this maximum output. Where the stomach can still deal with this maximum output operation is perhaps less likely to benefit the patient than where the power to deal with maximum output is diminished. In general, methods of assessment of suitability for operation are subjective and an objective approach should be a step forward. As yet the apparatus required is more suitable for research than day-to-day clinical application.

SUMMARY

In 51 patients with and without disease of the alimentary tract continuous records of gastric *p*H have been made for 24-hour periods. Each patient appeared to have a *p*H base-line.

No evidence was found of polarization, poisoning, mucus contamination, or burial in mucosal folds of the electrodes which might have led to disturbance of the record. Food and alkali caused only a brief change in *p*H.

The augmented histamine test was used in 19 of the patients, but the gastric contents were not aspirated. In this way an objective assessment of the patient's probable response to operation was made and found to be reliable in 12 patients.

Acknowledgements.—Miss Margaret Yule and Mr. Murray Ettle of the Department of Surgery, Queen's College, have been responsible for the illustrations and Messrs. Murray Ettle, Douglas Allan, and William Muir helped with the construction and maintenance of certain parts of the apparatus. The ward sisters and nurses gave invaluable help in recording times of meals, collecting specimens, and giving drugs.

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THE LATE RESULTS OF CLOSED RENAL INJURIES

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REVIEW of the world literature on closed renal injuries shows a considerable difference of opinion on their management, an abundance of information on their classification, but little on the long-term results of treatment. This paper presents the long-term results in 59 patients treated during the last 10 years in the United Bristol and Southmead General Hospitals.

The series was largely composed of young adult males. Road traffic accidents and football field injuries accounted for half the total cases in equal proportions. We found some difficulty in the classification of these patients and for convenience have divided them into two broad clinical groups:—

Clinically mild injuries	36
Those presenting with more severe injuries	23
	59

All patients presented with hæmaturia, and indeed this was often the only symptom in mild injuries. The injury was considered to be clinically more severe if the hæmaturia was profuse or prolonged, or if there were clinical evidence of shock or loin hæmatoma. Associated injuries were present in 12 cases, but in only 4 was the renal injury not the major problem; in 1 of these a ruptured spleen was the presenting feature, and the other 3 patients died of multiple visceral injuries shortly after admission to hospital.

RADIOLOGICAL INVESTIGATIONS

All except 8 patients were investigated in the early stages by excretion urography, and in many this was repeated to assess progress. Retrograde pyelography was performed on 5 patients at a later stage. In the clinically mild injuries the excretion urography was normal in 19 of the 32 cases, whereas urography was abnormal in all patients of the clinically severe group.

Table I shows the principal radiological signs and their incidence in the two groups. In addition,

Table I.—PRINCIPAL RADIOLOGICAL SIGNS AND THEIR INCIDENCE

	Mild Group	*Severe Group*
	32	19
Loss of the psoas shadow	6	9
Lumbar scoliosis	3	6
Delayed or absent function	4	7
Anatomical deformity of the caliceal pattern	4	11
Extravasation of contrast medium	2	5

5 of the clinically mild cases showed gross hydronephrosis on the side of the injury at first examination and 1 showed changes suggestive of chronic pyelonephritis in the contralateral kidney. Of the clinically severe group 1 case showed on the side of the injury a large hydronephrotic ectopic pelvic kidney, with a dilated extrarenal pelvis (John, 1960), and in 3 others the contralateral kidney was congenitally absent, hypoplastic, and hydronephrotic respectively.

MANAGEMENT

In the mild group immediate exploration was never seriously considered. Interval nephrectomy was performed on 4 of 5 patients with hydronephrosis for reasons not directly attributable to the injury.

In the severe group 3 patients died shortly after admission from multiple visceral injuries, 1 having undergone total nephrectomy. One patient underwent laparotomy and after splenectomy a laceration of the lower pole of the left kidney was palpated, but

no further operative treatment was performed. Total nephrectomy was carried out on 2 patients, one with hydronephrotic ectopic kidney and one with a severely damaged kidney, the contralateral kidney being hypoplastic. Partial nephrectomy was performed on the patient with severe injury to his only kidney.

The remainder were treated conservatively although in several cases exploration was urgently considered.

FIG. 221.—Solitary kidney with large staghorn calculus and hydronephrosis.

Apart from the treatment of shock the conservative management included complete bed-rest, copious oral fluids, and antibiotic cover. The provision of antibiotic cover for patients with renal injury is considered to be standard practice, but many of these patients apparently did not receive this therapy. Seven patients had urinary infection during the course of their illness, and it is interesting that 3 of these had pre-existing renal abnormalities—2 with hydronephrosis and 1 with a duplex kidney. No patient developed perinephric abscess.

The length and severity of the hæmaturia did not appear to bear any relationship to the severity of the injury, or to the subsequent course of the patient.

Method.—The long-term results were assessed from the clinical history of residual symptoms, a record of the blood-pressure, and excretion urography. We were unable to include, for various reasons, 12 of the mild group and 1 of the severe group in the follow-up, but all had relatively trivial initial symptoms and fortunately in all 12 mild cases the initial radiological examination was normal, and in the severe case lumbar scoliosis was the only abnormality.

Results.—The majority of the patients were symptom-free at the time of the follow-up which varied from 9 months to 8 years. Five patients were dead: 3 shortly after their injuries, 1 from an unrelated cerebral thrombosis, and 1 from failure of his hypoplastic kidney. Recurrent attacks of urinary infection with calculus formation have followed partial nephrectomy in the patient with a solitary kidney.

Whereas post-traumatic renal hypertension has been described, we found no evidence of this complication in our series.

RADIOGRAPHIC FINDINGS

(*Table II*)

Of the 24 cases in the mild group followed up, 11 showed normal radiographic appearances, initially and at subsequent examination, and 5 had a hydronephrosis not attributable to the injury. The remaining 8 cases showed initial radiographic abnormalities, but 5 of these had returned to normal at subsequent examination. One case showed only loss of the psoas shadow and lumbar scoliosis which later returned to normal, but 4 showing changes suggestive of caliceal rupture also returned to normal. In the remaining 3 cases, which all showed caliceal rupture initially, 1 now shows a caliceal diverticulum, 1 has an absent lower calix with shrinkage of the lower pole parenchyma, and the third has distortion of the lower pole calices.

FIG. 222.—Non-functioning upper pole of the left kidney with cicatricial incisura of outer border and calcification of renal parenchyma.

FIG. 223.—Retrograde pyelogram showing small scarred calicopelvic system.

Of the 16 patients with more severe initial injuries who attended for further excretion urography, 10 had returned to normal, and these included 4 in which the initial examination demonstrated definite rupture of one or more calices with extravasation of contrast medium. The remaining 6 had all initially shown radiographic evidence of caliceal rupture.

In the patient with the severe injury to his solitary kidney, who underwent partial nephrectomy, long-standing infection and sinus formation have culminated in a large staghorn calculus and diminished renal function (*Fig.* 221). Calcification was found in 3 patients: in 1 at the site of the previous perirenal hæmatoma, and in 2 at the site of the original caliceal rupture. In 1 patient there is dilatation of the upper calix with good function, presumably due to scarring

Table II.—RADIOGRAPHIC FINDINGS

Mild Group: 24 cases followed up
- 11 Normal
- 5 Hydronephrosis
- 8 Abnormal { 5 Normal; 3 Residual changes

Severe Group: 16 cases followed up
- 16 Abnormal { 10 Normal; 6 Residual changes

and resultant narrowing of the stem of the major calix. Localized caliceal dilatation was seen at an intermediate stage in 2 patients, both involving the upper poles in kidneys which had sustained lower pole ruptures: one has returned to normal, but the other has since become non-functioning (*Fig.* 222). In 1 patient the previously lacerated kidney was found to be small, presumably replaced by scar tissue, and completely functionless (*Fig.* 223).

DISCUSSION

Our studies indicate that where the initial radiological examination shows no evidence of caliceal rupture, whether the patient has a clinically mild or severe injury, the prognosis and long-term results are uniformly good. On the other hand, where a caliceal rupture was shown radiologically, our results show that in almost half the cases some permanent structural abnormality persists, although nearly all show good renal function and all are symptom-free and normotensive. In retrospect, however, we could find no features, either clinical or radiological, which would serve to predict the subsequent course of such a patient.

In our series urgent exploration of the kidney was performed in only 5 patients, and 3 of these had total and 1 partial nephrectomy. In these patients the indication for operative treatment was a deterioration in clinical condition due to severe sustained hæmorrhage uncontrolled by conservative methods. Later, elective nephrectomy was carried out on 4 of 5 patients in whom a pre-existent hydronephrosis came to light as a result of minor trauma. Speakers at the Annual Meeting of the American Urological Association (1949) stated that surgical intervention was commonly indicated in renal injuries, that any case having grossly bloody urine after twenty-four hours should be operated upon, and doubted whether there was any place any more for expectant treatment in handling renal injuries. Sargent and Marquardt (1950), while quoting these views themselves, adopt a much more conservative outlook, and in their series all patients with minor injuries were treated expectantly, and only 8 of 86 patients with more severe injuries had immediate operation. However, 9 of those remaining developed late sequelæ including 4 cases of perinephric abscess, most of these then being surgically explored. Our results agree with their findings in respect of the need for immediate operation, for which the only indication appears to be continued deterioration due to uncontrolled bleeding, but the complications they experienced in conservative management, including perinephric abscess, renal hypertension, and pseudo-hydronephrosis, were notably absent in our series. Badenoch (1950), in a series of 46 patients, operated upon 1 immediately and on 3 others within three weeks, while Ferrier and Knigge (1943) found operation necessary in 12 cases of a series of 93, but the long-term results in those treated expectantly are not mentioned.

Interval exploration was not undertaken in our series except for those cases of pre-existing hydronephrosis. Borham (1960) has suggested that late exploration is indicated in those patients developing dilatation of a previously uninjured calix because of the risk of sepsis, deterioration in function, or hypertension. In our 3 cases, 1 has returned to normal, 1 has normal function, and 1 has since become non-functioning, but all 3 are symptom-free with a normal blood-pressure, and we do not consider operation is justified. The patient with a small scarred non-functioning kidney is also symptom-free and normotensive.

Immediate excretion urography appears to be essential in all cases, provided the initial shock has been corrected and the blood-pressure is within normal limits. The principal value of this examination is the demonstration of a normally-functioning contralateral kidney. It will serve to confirm the presence of renal injury, and, although the extent of the damage may also be demonstrated, we consider this to be of secondary importance at this stage. Where the clinical condition precludes excretion urography, the presence of a normal contralateral kidney can sometimes be shown in a plain film of the renal tract, and in cases surgically explored, this must be confirmed by palpation across the peritoneal cavity. In this series the contralateral kidney was abnormal or absent in 4 patients who had suffered mild or severe renal injury. One of these clearly exemplifies the problems which may be encountered and will be briefly described.

Case.—A 17-year-old young man was injured while playing football and after resting for ten minutes returned to play for the remainder of the game. He was later admitted to hospital in a shocked condition with tachycardia, B.P. 60 systolic, and a mass in the right loin. After resuscitation for some hours, operation was performed and a disorganized right kidney had to be removed. The left kidney was palpated and found to be small and apparently hypoplastic. His post-operative condition appeared satisfactory, but his blood-urea, estimated one week after operation, was 250 mg. per cent. He progressed well but his blood-urea fell very little and six weeks later he developed increasing left loin pain and muscular rigidity. Cystoscopy was performed, followed by a left nephrostomy. When exposed, the kidney was found to be tense, thin, lobulated, atrophic, and hydronephrotic. His general condition continued to be satisfactory, the nephrostomy functioned adequately, but his blood-urea remained elevated and his hæmoglobin low. His condition very slowly deteriorated and he died 9 months after his original injury.

Retrograde pyelography is carried out in some centres (Jones, 1955; Orkin, 1950), often at an early stage in order to demonstrate the exact anatomy

of the injured kidney. This was not the practice in this series and we feel it has a limited value in view of the fact that many with severe renal lacerations subsequently return to normal. Moreover, the very real risk of sepsis in a large hæmatoma is a complication to be considered, and the 5 per cent incidence of perinephric abscess in centres where retrograde pyelography is commonplace tends to confirm our view. We have been impressed by the capacity of the kidney to return to normal function even after severe injuries. The following case serves to illustrate this point:—

FIG. 224.—Rupture of the lower pole of the left kidney with extravasation of contrast medium.

FIG. 225.—Left kidney showing complete return to normal appearances. (Same case as *Fig.* 224.)

Case.—An 8-year-old boy was reported missing from home and was found some three hours later to have fallen through the glass roof of a garage across the bonnet of a car 15 ft. below. He was gravely shocked and in fact appeared moribund, but after resuscitation his condition improved, hæmaturia occurred, and there was clinical evidence of intraperitoneal hæmorrhage consistent with splenic rupture. At laparotomy, a ruptured spleen was removed and the left kidney was palpated and found to have an extensive deep laceration of the lower pole. In view of the patient's general condition the kidney injury was treated expectantly. His post-operative recovery was uneventful, hæmaturia lasting only ten days, and excretion urography confirmed lower pole rupture with extravasation (*Fig.* 224). At the time of our follow-up three years later, the patient was symptom-free, normotensive, and the left kidney appeared completely normal (*Fig.* 225).

We consider that if initial excretion urography be normal, further radiological investigation in the absence of symptoms is unnecessary. If the initial examination is unsatisfactory we consider it advisable to repeat the examination before discharge of the patient from hospital. No follow-up examination is necessary for those with normal urographic findings at this stage. We would suggest that in those with radiological evidence of renal damage, further clinical and radiological review at a later date is indicated.

SUMMARY

A late follow-up in 59 cases of closed renal injury shows a uniformly good sequel in all the mild cases, and most of the severe cases treated conservatively. A high incidence of pathological kidney (pre-existing hydronephrosis) was noted in the mild group.

The management of closed renal injuries is discussed with particular reference to special investigations of both the injured and uninjured kidney.

Acknowledgements.—The authors wish to thank Dr. J. H. Middlemiss for his encouragement and advice, and the Consultant staff of the United Bristol and Southmead General Hospitals for permission to investigate patients under their care. We are indebted to Mr. W. Sweet for the illustrations.

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PALPATION OF THE LOWER COMMON BILE-DUCT AND THE HEAD OF PANCREAS

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PALPATION and exploration of the common bile-duct, particularly in its lower portion, have long been a perplexing problem to the young surgeon: nor have modern advances in operative cholangiography entirely solved this difficulty. Lytle (1959) has described very fully the common bile-duct groove in the back of the pancreas and illustrates palpation of the lower end of the duct from the lateral side of the duodenum, the examining fingers passing behind the duodenum and the head of pancreas, the thumb in front.

A different method of palpation of the lower end of the common bile-duct has been practised and taught for many years by Mr. T. McW. Millar, and because this method is not widely known, is simple to practise, and does not appear to have been described hitherto, it is illustrated below.

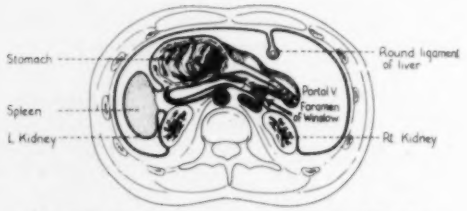

FIG. 226.—Cross-section of body at foramen of Winslow. (*After Hertzler*, 1919.)

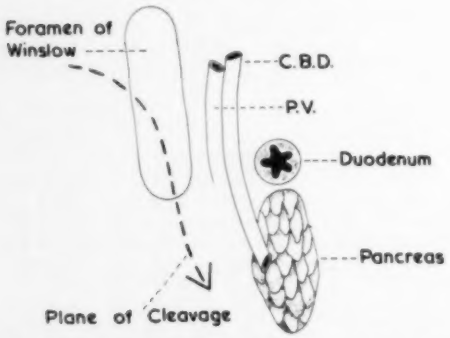

FIG. 228.—Plane of cleavage for palpation of lower common bile-duct.

ANATOMY

The foramen of Winslow is bounded in front by the important structures in the free edge of the lesser omentum. The inferior edge of the foramen is continuous with the right boundary of the lesser sac (*Fig.* 226). If the peritoneum constituting the right boundary of the lesser sac immediately adjacent to the foramen of Winslow is broken through by a finger and that finger then passed distally and to the right, it enters a plane behind the first part of duodenum, the hepatic artery, the portal vein, the common bile-duct, and the head of pancreas, and in front of the inferior vena cava; this plane is avascular.

THE MANŒUVRE IN SURGERY

At operation when palpation of the common bile-duct is desired, the fingers (usually the index and middle) of the operator's left hand are first passed into the foramen of Winslow. With the thumb in front of the lesser omentum the supra-duodenal portion of the biliary passages is now palpated between fingers and thumb (*Fig.* 227).

FIG. 227.—Initial position for palpation of common bile-duct.

FIG. 229.—Final position for palpation of common bile-duct.

The surgeon's left hand is now rotated about an axis through the metacarpophalangeal joints and with the index and middle fingers in the coronal plane. In this way the middle finger impinges on the right boundary of the lesser sac just to the left of the foramen of Winslow and, with gently increasing

pressure, splits the peritoneum (*Fig.* 228). The examining fingers can then be advanced downwards and to the right behind the head of pancreas to the ampulla of Vater at the medial border of the duodenum; with the thumb still on the anterior aspect of the head of pancreas the lower end of the common bile-duct and the head of pancreas can be palpated between finger and thumb (*Fig.* 229).

There remains a peritoneal edge on the lower margin of the foramen of Winslow which is unbroken. The manœuvre as described above has been used on countless occasions to examine the common bile-duct and has never given rise to difficulty. Bleeding does not occur. The manœuvre may be supplemented if so desired by palpation of the duct against a bougie within the lumen of the duct.

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MAMMARY-CORONARY ANASTOMOSES

A SHORT HISTOLOGICAL STUDY

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VINEBERG (1946) and Vineberg and Jewett (1947) first demonstrated that the internal mammary artery implanted into the anterior wall of the left ventricle remained patent, and formed anastomoses with adjacent branches of the left coronary artery. This interesting biological phenomenon stimulated much subsequent research into the method as a means of augmenting the blood-supply to a heart rendered ischæmic by coronary atheroma.

Among those who have attempted to repeat his experiment there has been some conflict of opinion about the validity of Vineberg's claims, and in particular about the frequency and permanence of the anastomoses. For this reason an experimental appraisal of Vineberg's work was undertaken in this Unit by Professor R. S. Pilcher, and the histological study reported here is part of this work.

MATERIAL AND METHODS

The material was obtained from 4 mongrel dogs of from 10 to 19 kg. in weight, whose left internal mammary artery had been implanted into the myocardium of the left ventricle by a technique modified slightly from Vineberg's original method in that the artery was mobilized almost to its origin from the subclavian, and several side holes were made in the artery prior to implantation. This was done either by opening ligated side branches or by making new holes in the arterial wall.

Three animals also underwent ligation of the anterior descending branch of the left coronary artery; 1 at the same time as implantation, and 2 eight weeks after. Two animals were sacrificed at sixteen weeks and 2 at twenty-five weeks, flow measurements on the internal mammary artery being made in each case at the terminal operation (*Table I*).

Since the intention in this particular part of the investigation was to demonstrate histologically any anastomosis between the implant and the coronary vessels, the above animals were selected from the main series as being those with the largest back-bleed from the cardiac end of the transected implant. Thus only animals with a minimum back-bleed of 2·5 ml. per min. were used, as by inference these had developed the best anastomoses.

After removal of the heart from the thorax the implant was cannulated, perfused with normal saline and then with Schlesinger's injection mass (1957) at 120 mm. Hg pressure, and finally unrolled by the technique of Schlesinger (1938). The entire heart was then fixed in formol saline and a block of tissue comprising the area of left ventricular wall with its implant removed. This block was then divided into three parts (top, middle, and bottom), and serial sections of each part were cut at 10 μ so that the entire length of the implant was sectioned.

Table I.—DURATION AND NATURE OF EXPERIMENTS, WITH BACK-FLOW FROM CARDIAC END OF IMPLANTED ARTERY

No.	OPERATION	DATE OF TERMINATION	BACK-BLEED/MIN.
8/58	I.M.A. implant	16 wk.	2·5 ml.
24/58	I.M.A. implant A.D.A. ligation at 8 wk.	25 wk.	4·3 ml.
26/58	I.M.A. implant A.D.A. ligation at 8 wk.	25 wk.	5·2 ml.
33/58	I.M.A. implant and simultaneous A.D.A. ligation	16 wk.	4·3 ml.

I.M.A. = Internal mammary artery.
A.D.A. = Anterior descending branch of left coronary artery.

Every fourth section was stained with Weigert's iron hæmatoxylin and van Gieson, intermediate sections being stained if necessary. Further sections were stained with Weigert's elastic, and normal internal mammary arteries prepared with the same stains, as controls.

RESULTS

General Histology of Implanted Arteries.—Compared with the normal the implanted artery showed changes which were conspicuous in the intima and to a lesser extent in the media.

1. *Intimal Changes.*—A constant finding was thickening of the intima by cellular tissue, containing mature and recent collagen, and elastic tissue, and lined by endothelium. There was variation in the degree of intimal thickening from implant to implant, and also within each individual implant. Two implants showed progressive narrowing of the lumen to the distal end, but in the other two this uniformity was not found (*Table II*).

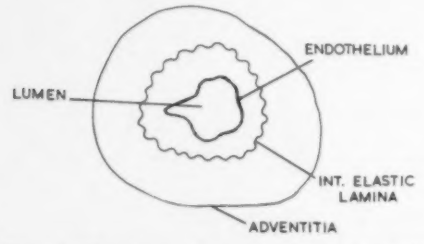

FIG. 230.—Transverse section of implant to show limiting areas used in calculating lumen sizes.

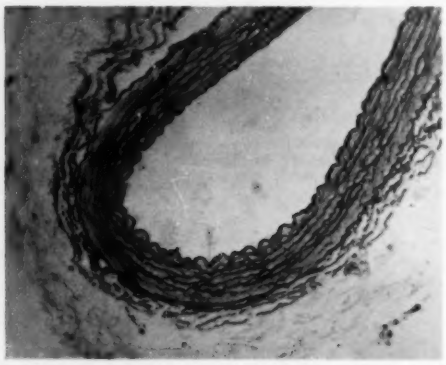

FIG. 231.—Normal internal mammary artery showing elastic of internal elastic lamina and media (Weigert's elastic). (× 93.)

The area of the arterial lumen at termination was expressed as a percentage of the original lumen. This was calculated by projecting stained sections of the implant on to plain paper with a camera lucida, and weighing first the total area of paper enclosed by the internal elastic lamina, and then the area enclosed by the endothelium (*Fig.* 230).

The artery in its extra-cardiac course showed little intimal thickening, but this became more marked as soon as the artery entered the tunnel in each case. One implant (8/58) was very narrowed in the middle of its course, but in the upper part of the bottom block the lumen was only 50 per cent occluded. Immediately after the only side branch was given off the lumen narrowed rapidly again and soon became completely obliterated. This rapid narrowing beyond the last branch was noted in 3 of the implants, and in the other (26/58), where no side branches were demonstrated, the vessel was still widely patent in the last section.

Table II also shows that the degree of intimal thickening is such that in the intramural portion of 3 implants the lumen was maximally only two-thirds patent. The effective volume of blood which can be carried by the implant must therefore be very considerably reduced.

Table II.—LUMEN SIZE AT TERMINATION OF EXPERIMENT EXPRESSED AS A PERCENTAGE OF ORIGINAL LUMEN; AND AVERAGE LUMEN RELATED TO BACK-FLOW

No.	LUMEN AS PERCENTAGE OF ORIGINAL			AVERAGE LUMEN	BACK-FLOW MIN.
	Top	Middle	Bottom		
8/58	69	10	50	43	2·5 ml.
24/58	59	22	10	30	4·3 ml.
26/58	61	62	70	64	5·2 ml.
33/58	91	65	13	56	4·3 ml.

2. *Media.*—This shows no muscular hypertrophy but a mild increase in collagen, and some increase and thickening of the normal elastic component (*Figs.* 231, 232). The general appearance suggests a slow atrophy of muscle in the implanted artery, and in 26/58, where the artery was widely patent at its distal end, the muscle was gradually replaced by mature collagen around a space lined by endothelium, until no muscle remained.

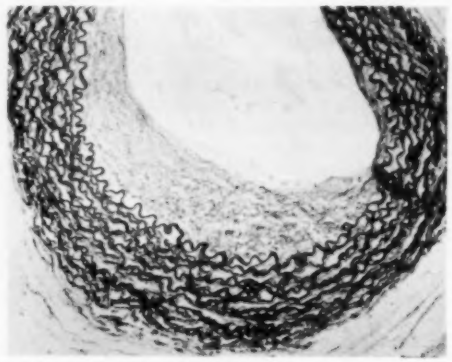

FIG. 232.—Implanted internal mammary artery showing thickening and increase of elastic components (Weigert's elastic). (× 93.)

3. *Adventitia.*—This was apparently normal, and became continuous with the fibrous tissue present around each implant. It contained a few elastic fibres, but no vasa vasorum were visible.

The fibrous tissue around the implant was marked in 2 cases (8/58 and 33/58), being due to infarction in the latter. In no case, however, was there any indication that the fibrosis was strangling or compressing either the implant or its branches. There were many muscular vessels present in the fibrous tissue, and it appeared in fact that these were of larger diameter when the fibrosis was most marked.

The surrounding fibrosis represents the only reaction to the implant, a cellular response being conspicuously absent except at the ends of the tunnel where silk ligatures were still present.

Branches and Anastomoses.—Side branches of the internal mammary artery were present in 3 of the 4 implants examined. The fourth showed no side branch but the vessel was still widely patent at the distal end of the tunnel (26/58). *Table III* shows that of the four branches found, three arose from the distal third of each of the implants. In 33/58, in which the only branch found arose from the distal end of the middle third, the implant was still widely patent at the level of the lowest section, and a further branch cannot be excluded.

Table III.—Number of Branches found by Serial Section, and Position in Tunnel Related to Number of Openings made at Operation

No.	Back-flow	Holes at Implantation	Patent Branches from		
			Top	Middle	Bottom
8/58	2·5 ml.	4	0	0	1
24/58	4·3 ml.	Several	0	0	2
26/58	5·2 ml.	3	0	0	0?
33/58	4·3 ml.	5	0	1	0?

It can also be seen that in no case does the number of branches correspond with the number of side holes made at the time of implantation. In only 1 animal (24/58) was there evidence of a thrombosed side branch. This occurred at the lower end of the middle block and was the only evidence found of a previous branch. It would appear that some branches regress and become incorporated in the arterial wall.

The side branches proper were all normally differentiated muscular vessels with a well-defined internal elastic lamina, in continuity with that in the implant (*Fig.* 233). In each case anastomoses could be demonstrated between these side branches and the muscular vessels running parallel to the implant in the fibrous tissue (*Fig.* 234 A–E).

It was not possible to demonstrate the actual union of vessels derived from the implant with definite branches of the coronary arteries, although this can be inferred from the way the coronary vessels had completely filled with Schlesinger's Mass injected via the implant.

The largest vessels were situated in the fibrous tissue close to the implant, but still smaller muscular vessels could be traced out into the myocardium. The finer components of the injection mass could also be shown in capillaries between the muscle-fibres, and also in spaces where no definite endothelium was demonstrable (*Fig.* 235).

The fact that Schlesinger's Mass will mostly not pass through vessels less than 40 μ diameter suggests an anastomosis of at least this dimension, but this was not demonstrable in these sections.

The new vessels showed no evidence of regression or thrombosis even at twenty-five weeks after implantation.

The serial sections did not reveal evidence of vasa vasorum, particularly in those cases where the adventitia was still distinguishable from the surrounding fibrosis, and the new branches running alongside the implant were too far removed from it to be merely enlarged vasa vasorum.

DISCUSSION

The work of Vineberg and his co-workers (Vineberg, 1946; Vineberg and Jewett, 1947; Vineberg and Niloff, 1950; Benscombe and Vineberg, 1953; Vineberg and Buller, 1955; Vineberg, 1958); Glenn, Holswade, and Gore (1950); Glenn and Beal (1950); Bakst, Maniglia, Adam, and Bailey (1955); Maniglia and Bakst (1956); and Bellman and Frank

Fig. 233.—Showing continuity of coats with prominent internal elastic lamina in implant and branch (Weigert's elastic). (× 47.)

(1958), amongst others, has demonstrated convincingly that a systemic artery implanted in the myocardium is capable of forming anastomoses with branches of the coronary arteries. There is, however, less uniformity of opinion as to the duration of these anastomoses, the factors which influence their formation, or the nature and cause of the intimal thickening which is such a constant feature, at least of internal mammary implants (Fuquay, Carey, Dahl, and Grindlay, 1958).

Glenn and others (1950) and Glenn and Beal (1950) were of the opinion that the new vessels which formed from the implant resembled granulation tissue, and as such tended to regress or disappear after about six weeks. Bakst and others (1955) and Maniglia and Bakst (1956) agreed with this and thought that the vessels were the end-result of the inflammatory reaction produced after implantation. Certainly the new vessels must develop originally from capillaries in the granulation tissue which forms after implantation and under the influence of pressure and flow (Clark and Clark, 1934), and possibly substances derived from damaged cells, fully differentiated arterioles are formed. Once these are carrying blood to the myocardium there is no reason why they should regress, and the present investigation holds no support for the regression theory. After twenty-five weeks muscular vessels connected with the implant were still present, and capable of carrying blood into the myocardium, although organization around them had proceeded to the formation of dense fibrous tissue. Vineberg (1958) has shown that the longer the implant is left before termination, the greater the area of myocardium supplied from it.

The factors influencing the formation of side branches are still obscure. This small series shows that the making of a side hole in the internal mammary artery, which bleeds prior to implantation, is no

guarantee that a side branch will develop at that site. The continuity of coats and relative sizes of branch and implant, however, suggest that the branches found did develop from pre-existing intercostal branches. There is no histological evidence to support Bellman and Frank's (1958) contention that the side branches arose from vasa vasorum.

A further point of interest concerning the branches is that most of these were found in the distal part of the tunnel. Bellman and Frank (1958) noted this in 90 per cent of cases and attempted to explain it on mechanical grounds, but it may well be that increased tissue damage caused by the placing of sutures at the distal end of the tunnel, or relative anoxia towards the apex of the heart after anterior descending artery ligation, may stimulate new vessel formation and differentiation to a greater degree at the lower end of the tunnel than elsewhere. That ischæmia does influence the implant is shown by Vineberg and Buller (1955), and later by Pearl, Joseph, and Citret (1959), when partial occlusion or ligation of the anterior descending artery increased the rate of mammary–coronary anastomosis from 46 per cent to 71 per cent, and from 11 per cent to 85 per cent respectively. In our experiments it has been noted that good anastomoses developed in statistically significant numbers in those implants where it had been necessary to withdraw and re-implant the artery immediately afterwards for technical reasons. It is tempting to suggest that this increased success rate may have been due to increased damage to the myocardium.

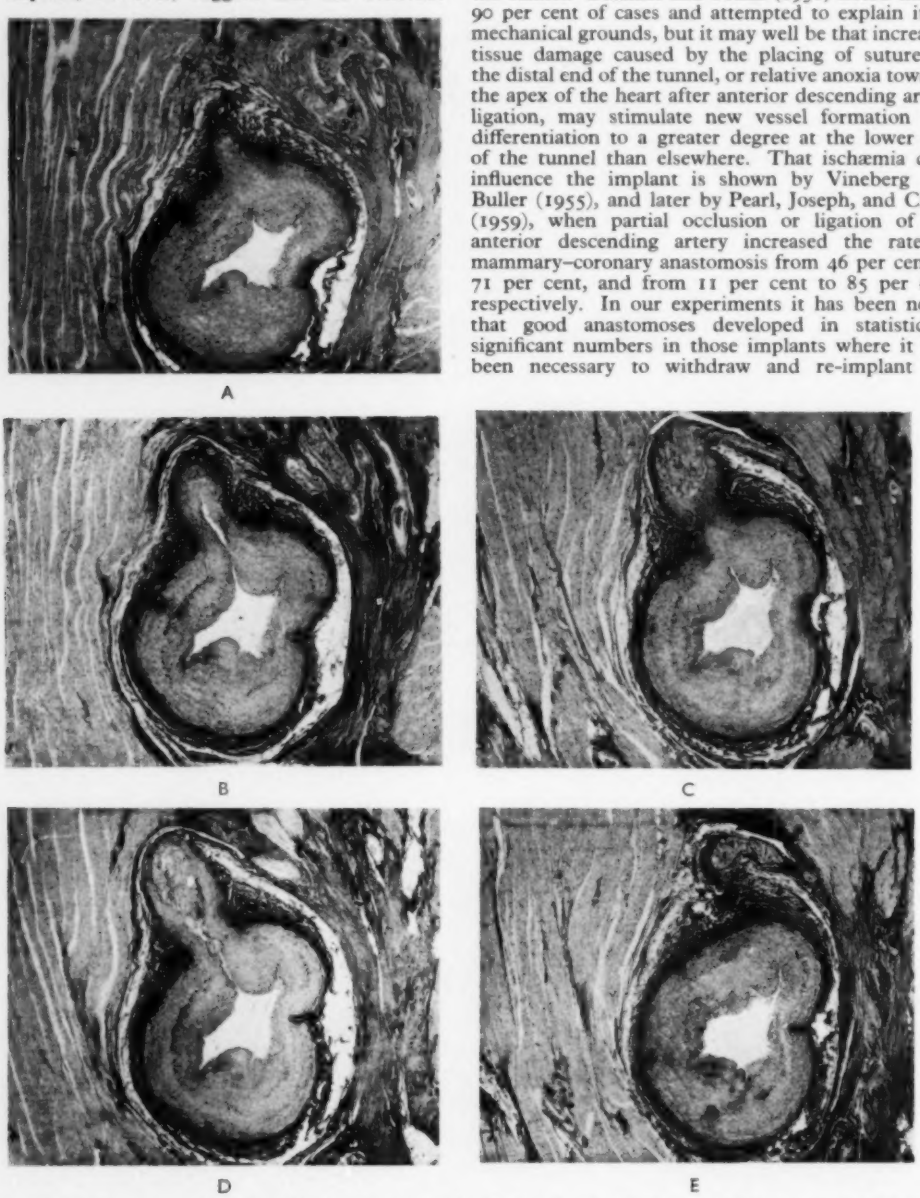

FIG. 234.—A–E, Transverse serial sections of implanted artery showing the formation of a side branch with injection mass in its lumen, which joins with adjacent vessels passing to the myocardium. Note the greatly thickening intima. (× 24.)

INTIMAL THICKENING

This has been noted as a constant feature in all implants studied, and the degree of occlusion is always marked in unfavourable reports (Patt, Goldberg, Clift, and Lourvanij, 1958). Various causal factors have been incriminated, among which are degenerative changes due to nutritional insufficiency

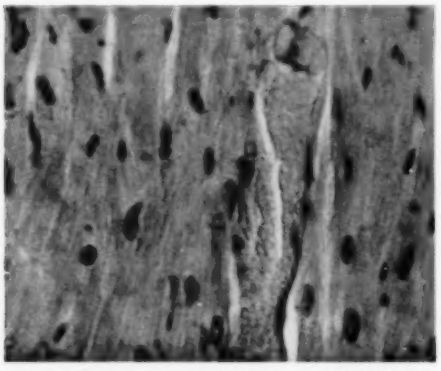

FIG. 235.—High-power view of injection material within myocardial sinusoid. (× 532.)

while within the tunnel; trauma to the implanted part of the artery; or thrombosis due to trauma or stasis, with or without subsequent recanalization.

In the 4 implants studied it was impossible to prove or disprove the last hypothesis, although there was no evidence of recanalization of the occluding mass in any section. Moritz and Oldt (1937) describe intimal thickening due to three causes: (1) hypertension; (2) adjacent chronic inflammation (cf., Doniach, 1949); and (3) an adaptive reaction due to reduction of the capillary bed normally supplied by an artery. The first two causes are not applicable to the present study, but the intimal changes resemble those seen in arteries of the ovary or uterus during involution, or in the hydronephrotic kidney, without hypertension.

In these implantation experiments it is apparent that the volume of blood carried by the internal mammary artery is attempting to enter a small capillary bed, at least immediately after implantation. The intima may react to the reduced out-flow by proliferating and thus reducing the blood-flow. Once equality between in-flow and out-flow has been reached, the proliferation ceases, and in none of the implants examined was the thickening an actively continuing process. Support for this idea of reduced out-flow is given by the work of Nachlas, Myers, Solomon, Kelly, and Seligman (1958) who found that 100 per cent of implanted carotid arteries remained patent when their distal ends were allowed to bleed into the right ventricular cavity, but that only 11 per cent remained patent when the distal end was closed.

SUMMARY

A histological study was made of a small series of 4 implants of the left internal mammary artery into the left ventricular myocardium.

Branching of the implant was observed in 3 implants and inferred in the other, with the formation of well-differentiated arteries which extended into the myocardium and anastomosed with vessels probably derived from the left coronary artery. These anastomotic vessels showed no tendency to regression up to twenty-five weeks.

Variable occlusion of the lumen of the implant by thickening of the intima was noted in all cases, and factors affecting this, and branching of the implant are discussed.

Acknowledgements.—I wish to thank Professor R. S. Pilcher for his help, and for allowing me to study material obtained from animals upon which he had operated; and also Mr. P. G. Luther and others for technical assistance.

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INGUINAL HERNIA AND THE ILIOPECTINEAL LINE

DEFICIENCY OF THE INSERTION OF THE CONJOINT TENDON AS A FACTOR IN THE AETIOLOGY, WITH AN ACCOUNT OF AN OPERATION FOR ITS CORRECTION AND A REPORT OF 200 CASES

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It might be thought that everything possible has been said and written about inguinal hernia, but allowance is not always made for variations in the anatomical arrangements of the inguinal canal and particularly that of the conjoint tendon. The extent of its insertion into the iliopectineal line appears to be of paramount importance in the aetiology of all large indirect and direct hernia cases, and the following is an account of a method of examination to ascertain this, and of an operation for restoration in defective cases, along with a review of over 200 cases personally operated upon (aged 2–76 years).

ANATOMY

The Iliopectineal Line and Insertion of the Conjoint Tendon.—Having through the courtesy of the authorities of the National Library of Wales studied various anatomy books published since 1854, I have found much discrepancy in the details of the anatomy of the posterior inguinal wall in general and the relationships and attachments to the iliopectineal line in particular. The following paragraphs are condensed from some of the anatomy books studied:—

The lowest fibres of the internal oblique are inserted into the pectineal line of the pubis in common with those of the transversalis muscle. Hence the tendon of this insertion is called the 'conjoint tendon of the internal oblique and transversalis'. This structure corresponds with the external abdominal ring, and forms a protection to what would otherwise be a weak point in the abdomen. Sometimes the tendon is insufficient to resist the pressure from within and is forced through the external ring; it then forms the distinctive covering of direct inguinal hernia. (Wilson, 1854.)

Obliquus abdominis internus muscle—the fibres from Poupart's ligament, usually paler than the rest, arch downwards and inwards over the spermatic cord, or the round ligament of the uterus, and end in tendinous fibres common to them and the lower part of the transversalis muscle, and hence known as the conjoint tendon of these muscles; through the medium of this tendon they are attached to the front of the pubis, and for some distance along the iliopectineal line behind and to the outside of Gimbernat's ligament.

Transversalis abdominis muscle—lowest fibres curve downwards like the internal oblique and are inserted into the front of the pubis and into the pectineal line through the medium of the conjoint tendon. (Quain, 1876.)

The lowest fibres of the internal oblique and transversus coming from the inguinal ligament are connected, when it exists, with the tendinous expansion, which has been known in this country as the 'conjoint tendon'. (Quain, 1923.)

The Conjoint Tendon is the tendon that gives a common attachment to the fibres of the internal oblique and transversus muscles which arch across from the inguinal ligament. It gains attachment to (1) the pubic crest and (2) the medial portion of the pectineal line for 1 in. The tendon is formed principally by the transversus abdominis, which extends for 1 in. along the pectineal line, while the internal oblique only extends along that line for ½ in. (*Fig.* 236.)

The Interfoveolar Ligament.—The conjoint tendon is usually limited to the first inch of the medial portion of the pectineal line. Sometimes, however, the anterior aponeurosis of the transversus abdominis is prolonged laterally beyond the normal limit of the conjoint tendon in the form of a thin semilunar expansion which extends as far as the medial and lower parts of the deep inguinal ring and is attached inferiorly to the deep femoral arch. This semilunar expansion of the conjoint tendon is known as the 'interfoveolar ligament of Hesselbach'. Its concavity is directed laterally and, when present, it covers the lateral third of the floor of the inguinal triangle where it lies behind the spermatic cord and is closely connected with the subjacent fascia transversalis to which it imparts strength. The external oblique, internal oblique, and transversus abdominis muscles cross each other at different angles, the external oblique passing downwards and forwards, the internal oblique upwards and forwards, and the transversus horizontally forwards. They thus form an intricate lattice work that renders the abdominal wall very strong and is a powerful safeguard against hernial protrusions. (Jones, 1946.)

The Relation of Structures at the Medial Portion of the Pectineal Line.—The relation of structures from before backwards is as follows:—

1. The pubic lamina of the fascia lata, incorporated with which is the pectineal ligament (ligament of Cooper).
2. The pectineus.
3. Pectineal part of inguinal ligament.
4. The conjoint tendon.
5. The fascial transversalis and deep femoral arch.

The aponeurosis of the internal oblique is fused with the aponeurosis of the transversus which is deep to it. The conjoint aponeurosis is inserted in front into the lower part of the linea alba and below into the crest of the pubis in front of the rectus; and in the majority of male subjects *there is extension of the insertion laterally along the first inch or more of the pectineal line. This lateral extension may be altogether absent or it may involve only the aponeurosis of the transversus*, but when it is present it receives in most subjects the lowest fibres of the internal oblique and transversus and is known as the 'conjoint tendon' (Jones).

The conjoint tendon is the name given to the part of the fused aponeurosis of the transversus and internal oblique muscles which is inserted into the anterior end, about 1 in., of the pectineal line; the transversus forms the larger part of it. It is lateral to the rectus muscle and lies immediately deep to the superficial inguinal ring, and passes downwards to its pectineal insertion deep to the inguinal and lacunar ligaments. The spermatic cord or round ligament lies in front

of it before passing through the superficial ring. As already stated it varies greatly in size, and the dissector will find that often (*in 20 per cent of subjects*) *he is unable to define a conjoint tendon as here described.* It may be absent or only slightly developed, or replaced by a lateral extension of the tendon or origin of the rectus muscle; but sometimes, when it is well developed, the conjoint tendon itself is

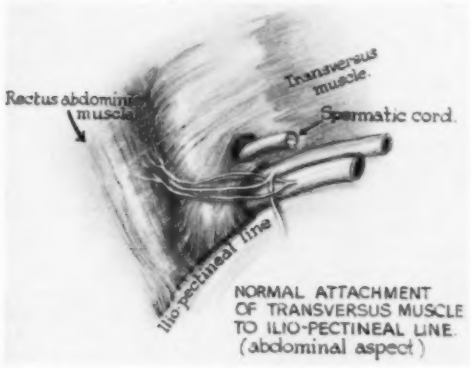


FIG. 236.—Normal attachment of transversus muscle to iliopectineal line (abdominal aspect).

FIG. 237.—Absence of attachment of transversus muscle to iliopectineal line as in large indirect or direct hernias.

continued farther laterally as a thin expansion which reaches the medial side of the deep abdominal ring so that then no interval is present between the lower border of transversus and the inguinal ligament (Walmsley, 1939).

Patey (1949) as a result of electrical stimulation of the internal oblique and transversus muscles in hernia cases makes a definite statement in his summary of the functional anatomy of inguinal hernia that the shutter-like mechanism of the conjoint tendon does not exist. Rains (1951), however, points out that the shutter action of conjoint tendon can only be demonstrated when the transversus and internal oblique muscles are attached to the iliopectineal line.

To realize the full importance of the attachment of the internal oblique and transversus muscles to the iliopectineal line, the posterior wall of the inguinal canal must be visualized from its intra-abdominal aspect. (*Fig.* 237.) The three fossæ so often described in text-books may for all practical purposes be regarded as two: the fossa lateral to the deep epigastric vessels as the congenital indirect hernial fossa; and the medial as the direct hernial fossa, either congenital or acquired—congenital when the muscle insertions are completely absent, and acquired when, although present to some degree, they are so thin and attenuated, as the result of increased intra-abdominal tension or strain, that any support to the posterior inguinal wall is gradually weakened and becomes useless.

The defect therefore shows itself in the first instance in either the medial or the lateral fossa, and so to prevent any recurrence the appropriate fossa must be strengthened.

Conclusions.—

1. The conjoint tendon is 80 per cent transversus and 20 per cent internal oblique and, under normal circumstances, the internal oblique insertion extends ¼–½ in. along the iliopectineal line, and the transversus muscle is inserted for 1 in. along the iliopectineal line. This is normal and occurs in 80 per cent of patients.

2. In 3 per cent of cases the conjoint tendon is attached to the rectus sheath alone and has no insertion into the iliopectineal line or pubic crest.

3. The insertions of the internal oblique and transversus muscles (conjoint tendon) vary enormously, and in 20 per cent they are attached only to the rectus sheath, pubic spine, and crest, and have no attachment whatsoever to the iliopectineal line.

4. The interfoveolar ligament may take the place of the conjoint tendon and spread laterally as a sheet of fascia supporting the fascia transversalis and usually joined to it, thus forming a fairly good support for the posterior wall of the inguinal canal.

5. The shutter action suggested by Keith occurs only when the muscles have their normal attachment to the iliopectineal line.

TREATMENT

Examination of the Patient.—Much of the anatomical arrangement can be ascertained by a careful examination of the patient when he lies on the examination couch and the inguinal regions are investigated. After inspection, and noting any impulse when the patient coughs, the examiner, standing on the left side of the patient, inserts his left index finger, palmar surface downwards, into the right external ring. If the iliopectineal line can be clearly and easily felt, the normal attachment of the transversus and internal oblique muscles cannot be present. If, however, a slight resistance is felt, yet the iliopectineal line is fairly clear, the presence of the interfoveolar ligament may be suspected. The degree of weakness present is in proportion to the extent of the insertion of the internal oblique and transversus muscles into the iliopectineal line.

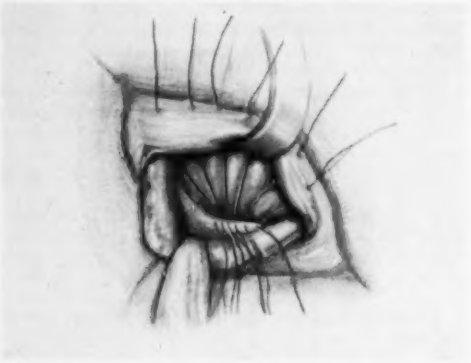
The left side should be examined from the patient's right side, and with the right index finger. The examination may be repeated on both sides with the patient standing. In some young men, without hernias, one finds that the normal insertions

of the conjoint tendon are not present and it is in these subjects I believe that when they reach middle age and suffer from increased intra-abdominal tension or indulge in work heavier than normal, direct hernias will develop. These factors should be borne in mind when patients are examined for the Services, or are about to undertake strenuous physical work as the compensation factor is bound to arise.

down and so the angle and level of our repair must be visualized as if the patient were standing up. Thus it is suggested that the conjoint tendon should not be brought anteriorly towards the inguinal ligament, but posteriorly to its normal insertion into the iliopectineal line, so that there is adequate support to the posterior wall of the inguinal canal inferiorly—where it is required most of all in the upright position. Under light anæsthesia it has been proved that when

FIG. 238.—Note distance between re-curved edge of Poupart's ligament and iliopectineal line.

FIG. 239.—Indirect and direct hernia with deep epigastric vessels between—two forceps on indirect sac and one on direct—to illustrate Huguet's manœuvre.



A

B

FIG. 240.—A, Line of sutures placed between conjoint tendon and iliopectineal line; B, Showing triangular gap in rectus sheath after tying sutures.

These anatomical variations have been found to be present at all ages when examination is carried out on the operating table. If present in young children with congenital indirect inguinal hernia, account should be taken of them so that, when their congenital indirect inguinal hernias are radically cured, steps are also taken to prevent the development of a direct hernia later in life.

Sir Heneage Ogilvie and others have always emphasized that the anatomy of the inguinal canal area should be reconstructed in as normal a fashion as possible and that the inguinal ligament (Poupart's) was never intended to be used as an anchor to which everything in the vicinity should be sutured.

Another factor which should be borne in mind is that we operate on the patient whilst he is lying

the patient coughs or strains the conjoint tendon contracts and thus becomes much closer to Poupart's ligament—but not the complete shutter action which occurs when the muscles are attached normally. It is obvious, therefore, that any implants such as fascial or nylon grafts joining the conjoint tendon to Poupart's ligament must become slack when the conjoint tendon contracts and when the support is needed. (*Fig.* 238.)

In congenital indirect inguinal hernias all that need be carried out is removal of the sac as high up as possible and narrowing of the internal ring if it has been dilated—*provided always that the internal oblique and transversus muscles are inserted normally into the iliopectineal line.* If these insertions are missing they must be replaced, otherwise a so-called

'recurrence' will take place in the form of a direct hernia.

In cases of large indirect inguinal hernias—the deficiency in the posterior inguinal wall giving rise also to a direct hernia—Huguet's manœuvre of increased traction on the indirect sac, after mobilization of the deep epigastric vessels, may be used, thus tightening up the peritoneum of the direct hernia. (*Fig.* 239.)

The Iliopectineal Line.—In addition to the relations already described there are other structures that must be borne in mind. Aberrant obturator vessels are frequently present and arise from the external iliac or occasionally the deep epigastric vessels. Another small artery and vein occasionally run along the iliopectineal line towards the pubic crest. These must be ligated medially and laterally within ¼ in. of the femoral vein before any sutures are placed in Astley Cooper's ligament, otherwise troublesome hæmorrhage or a hæmatoma may occur. This is very much easier to carry out if the transversalis fascia is divided prior to ligation of these vessels.

Lotheissen (1898) was the first to suture the conjoint tendon to Cooper's ligament. Koontz (1958) makes reference to this in a recent paper. McVay (1954) has carried out many operations based on the same principles with excellent results on all types of inguinal and femoral hernias.

Operation.—The incision, 3 in. to 4 in. long, is made 1 in. above and parallel to Poupart's ligament. The index finger is then placed in the external ring to lift up the external oblique aponeurosis, and a small incision is made in the line of the fibres of the external oblique ½ in. long, more towards the medial than the lateral side and 1 in. above the external ring. A blunt-ended scissors is then used to extend the incision laterally after freeing the external oblique from the contents of the cord and bearing in mind the position of the ilioinguinal nerve. The incision is prolonged downwards to the external ring. At this point it is noted that the edge of the external ring may be thicker than normal and an unusually strong, tough band runs along from it to be attached to the pubic crest, and sometimes the pubic spine. When this occurs the band is severed as far medially as possible and kept as a reminder when closing the external oblique and to reform the external ring as a final stage of the operation.

The external oblique flaps are cleared inferiorly to the re-curved edge of Poupart's ligament, and superomedially to the rectus sheath.

Congenital Indirect Inguinal Hernia.—The cord is examined and freed posteriorly by incising the cremasteric fascia along the upper border and the sac is cleared as high up as possible, a retractor being placed in the internal ring and drawn upwards and laterally, until the extraperitoneal fat is reached. The sac is opened and examined and occasionally twisted so that the peritoneum on either side of the base is taut. It is then ligated and the excess removed. I do not consider there is any necessity to transfix the sac and fix it to the overlying musculature. *Any lipoma must be removed as it may act as a wedge and form a weak point in the internal ring.* If the posterior wall of the inguinal canal is normal, as defined above, then all that remains, after restoring the internal abdominal ring to its normal size if any dilatation has taken place, is to suture the external oblique aponeurosis from superolaterally to inferomedially by a continuous No. 1 catgut suture. Prior to this, I insert two interrupted sutures to fix the band of the external oblique aponeurosis, to which a forceps was left attached, firmly to the pubic crest, producing a taut external ring.

Direct Hernia or Large Indirect Hernia.—The first thing to do is to define Astley Cooper's ligament or the iliopectineal line and to ascertain if there is any insertion of the conjoint tendon into it. Any abnormal blood-vessels are noted and ligated as described above. The position of the femoral vessels is noted, but they are not displayed. Any fatty processes in the region of the femoral canal are removed. The curving edge of the conjoint tendon is next defined, oftentimes with difficulty, as the fascia of the transversus muscle merges into the transversalis fascia. The transversalis fascia should always be incised to make certain of ligating any blood-vessels on the deep aspect of the iliopectineal line and to enable the operator to insert a finger into the preperitoneal space and separate the conjoint tendon away from the peritoneum when sutures are inserted. The edge of the conjoint tendon is then freed until the index finger can be placed for ½–1 in. underneath the whole length of the conjoint tendon as far as the internal ring laterally. In the majority of cases the sac of a direct hernia need not be opened, but it is advisable to clear away any excessive fat and adipose tissue, as emphasized by Professor Chester B. McVay in his paper on hernias. A small piece of gauze is then placed underneath the cord, clamped with a forceps, and used as a retractor to pull the cord inferiorly and laterally. A relaxing incision is next made in the rectus sheath approximately 2 in. long and ending within ¼ in. of the pubic crest with its upper end slightly more lateral than the lower. A small blood-vessel usually has to be tied in the upper two-thirds of the incision. Six thread sutures (Barber's 25) are next placed in the following manner: with straight forceps holding each end of the 1st, 3rd, and 5th; and curved forceps the 2nd, 4th, and 6th. (*Fig.* 240 A.) The first suture is then placed into the lacunar ligament, and on to Astley Cooper's ligament, through the adjacent portion of the conjoint tendon. When placing the sutures through Astley Cooper's ligament the left index finger should always be placed just medial to the femoral vein, to prevent any damage to that structure. Similarly when placing the sutures through the conjoint tendon, the left index finger should be placed underneath it, separating it from the peritoneum, and so preventing the needle from damaging the peritoneum. Care should be taken to include some of the rectus sheath in the suture, as if the suture is placed through muscle only it will tend to cut through. The needles used are round-bodied, semi-circular, ½–¾ in. with blunted points. The remaining sutures are placed fanwise—radiating from the medial first inch of the iliopectineal line to just above the internal ring—usually 2¼ in. Care is taken with the two most lateral sutures not to injure the deep epigastric vessels. The sutures are tied firmly in the same order as they were placed, starting medially. (*Fig.* 240 B.) Care is now taken that the ilioinguinal nerve is retracted downwards and not caught up in the sutures. One final suture (if necessary two) is

placed after the other sutures have been tied, between the fascia of the transversus, transversalis fascia, and the anterior femoral sheath (the latter picked up by forceps about ½–¾ in. from the recurved edge of Poupart's ligament, to ensure no damage to the femoral vessels). This last suture closes the aperture in front of the femoral sheath which prevents the possibility of a prevascular recurrence. It is not usually necessary to place any sutures in the relaxing incision which will now be seen to have become triangular in shape. The external oblique aponeurosis is sutured in the same manner as described in the indirect inguinal hernia operation, particular notice being paid to the two primary interrupted sutures.

In some cases of very large indirect and direct hernias the conjoint tendon as such may be attenuated to such an extent as to be almost non-existent, and the defect may have to be remedied by using a lattice darn with thread or fascial strips, nylon mesh, or something similar; but *in each case the area between the rectus sheath junction with the conjoint tendon down to the pubic crest and the iliopectineal line must be covered.*

Post-operative Treatment.—The following day patients are instructed to move their legs freely in bed with emphasis placed on plantar and dorsiflexion of the feet. Deep-breathing exercises are also encouraged. They are not allowed up until the sutures are removed on the eighth to tenth day and then they are instructed in leg and abdominal exercises by a physiotherapist. They are usually discharged on the fourteenth to fifteenth day.

CONCLUSIONS

The main point in this paper is to stress the importance of the iliopectineal line, and the degree of attachment to it of the conjoint tendon, the attachment being defective or completely absent in some cases. As the internal oblique muscle is normally attached for ½ in., and the transversus muscle for 1 in., any hernia repair to a weak posterior wall of the inguinal canal must take this into account and ensure that these muscles are replaced in as normal a position as possible.

In June 1958, with some colleagues, I examined approximately 80 patients whose ages ranged from 6 to 72 years, and whose hernia repairs based on the above considerations were done from four years to a few weeks previously. There was not a single recurrence among them. Some of the patients had had previous operations for the hernia and in 1 case two previous operations. This last case had recurred after a combined Bloodgood and Halsted operation performed six years previously. In every case at operation the main defect, i.e., the absence or inadequacy of the attachment of the internal oblique and transversus muscles to the iliopectineal line was noted. Only 4 of the patients written for did not come for examination, but it has since been found that all are healed soundly with no recurrence.

In October, 1959, I found 1 patient with a recurrence. He was very stout, 48 years of age, with chronic bronchitis, and had been operated on on July 11, 1957. At the next operation two lipomata were seen coming down through the internal ring with a thin peritoneal sac between them. The lipomata were removed and the sac ligated as usual. The original repair to the posterior wall of the inguinal canal was found to be firmly united and sound. The raw area over the rectus muscle from the relaxing incision was found to be completely healed over. The case was seen four months later, when the wound was soundly healed and there was no suggestion of any recurrence.

Since 1954, I have now carried out over 200 operations, using this method, on patients varying from 2 to 76 years of age. The results have been eminently satisfactory and it was only in the case of the patient quoted as having a lipoma coming down through the internal ring that there has been any possible recurrence.

Complications.—One double-sided hernia, a man of 62 years of age, died from pulmonary embolism on the sixth day. Another succumbed to coronary thrombosis whilst eating breakfast on the second post-operative morning. In 3 cases œdema of the scrotum occurred but, with proper support for the scrotum and testicles, with a suspensory bandage immediately after operation, this has now been obviated. One patient had post-operative pneumonia, from which he recovered. No severe sepsis occurred and the thread sutures gave no trouble.

SUMMARY

1. A defect, complete or partial, in the posterior wall of the inguinal canal, due to non- or deficient attachment of the internal oblique and transversus muscles to the medial portion of the iliopectineal line, is present in all direct and large indirect hernias. This can be ascertained by careful examination.

2. If the muscles are attached normally, neither a direct nor a large indirect hernia is likely to occur.

3. Two hundred consecutive patients, ages 2–76 years, have been followed up after operation and all except the 1 recurrent case are carrying out their normal work, are free from recurrence, and are no longer hernia-conscious.

4. The technique of the operation is described and is not difficult.

5. This operation is carried out for all direct and large indirect inguinal hernias as well as femoral hernias.

In cases of strangulated hernias I prefer to use catgut rather than non-absorbable sutures.

Acknowledgements.—My very grateful thanks are due to Professor Lambert Rogers, Mr. David Ioan-Jones, and Mr. Ivor Lewis for their very kind help and encouragement.

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A PENETRATING WOUND OF THE PALM WITH AN INDELIBLE PENCIL, PRESENTING AS A DIGITAL NEURITIS

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THE dangers of a penetrating injury with an indelible pencil have long been recognized and are well documented. Despite this it is felt that a further report is warranted, as these injuries are rare. Ferrier (1948) commented that the dangers of these injuries and similar conjunctival lesions were not generally recognized in this country although well known on the Continent and in America, and review of the literature, especially that in English, revealed that most of it was over ten years old and largely dealt with ophthalmic lesions. In text-books Cutler (1942) briefly touches on this topic and Bunnell (1956) deals thoroughly with it, but these texts are unlikely to be read systematically in the early days of post-graduate training. Among more general books Bailey and Love (1956) mention it, but in several other books widely used by postgraduate surgical students it is not described. Mason and Allen (1941) when reporting two cases said that the "characteristic chain of events which follows them warrants an occasional report of a case and justifies re-emphasis of their importance", and Kisner and Blake (1941) stressed that they are major injuries both from the physical and the economic standpoint. It would seem that unless a lesion such as this is occasionally reported there is a danger that the lessons learnt in the past may become forgotten and the very people most likely to be confronted with it will be inadequately prepared to deal with it efficiently.

The case here described is of interest because the absence of the usual signs of such an injury and the denial by the patient of its possibility led to delay in surgery.

HISTORICAL SURVEY

The destructive agent in indelible pencil injuries is methyl violet (penta-methyl para rose aniline), a basic dye related to aniline and combined with graphite and a binder, such as gum tragacanth, to form the pencil-lead. We are now aware that many of the derivatives of aniline have a harmful effect on living tissues. In addition to the specific lesion dealt with in this report, dermatologists have long known the association between aniline dyes and epithelial hyperplasia, whilst their carcinogenic effect on the bladder is well established. It was just over twenty years after the introduction of methyl violet commercially that the first report of an injury due to it appeared in the medical press. Thompson (1888) described the results of indelible pencil-lead falling into the conjunctival sac. Following this there were numerous reports from German ophthalmologists describing orbital lesions and noting the rapid diffusion of the dye to the anterior chamber of the eye resulting in a panophthalmitis and occasionally destruction of the eye. Kuwahra (1903) established that it was the dyestuff that was the necrotizing agent, whilst Vogt (1905) was able to show that the toxicity of these dyes was related to their alkalinity and suggested 4 per cent tannic acid as a neutralizing agent.

The first report of hand lesions came from Erdheim (1914) who did a great deal of experimental work and suggested that these dyes were toxic because of the affinity of the basic dye for the nucleic acid of the cell nucleus. Jerasalem (1923) discussed the need for amputation in digital lesions and Milch (1928) reporting one case said that the use of these pencils had been prohibited in German schools because of their dangerous nature. Puoz (1933) recorded the incidence of systemic malaise and gastro-intestinal upsets in about 20 per cent of cases, and this was followed up in experimental work by Gierlich (1935) showing the systemic uptake of the dye as a colourless substance that acted as a protoplasmic poison producing cloudy swelling and fatty degeneration in the liver.

Sharlet (1938) made an interesting observation of the development of a melanoma in an indelible pencil 'tattoo' and in one of the two cases recorded by Mason and Allen (1941) they took a skin biopsy at a second operation and found hyperplastic changes in the epithelium. The same article also gives an excellent bibliography of this subject.

Kisner and Blake (1941) experimentally implanted indelible pencil-lead into animals and found it to be non radio-opaque, which agreed with their clinical experience, and more recently Hosford and Smith (1952) reported the use of fluorescein in treating an ophthalmic lesion, describing the dye being precipitated out of the tissues and giving the chemistry of the reaction.

PATHOLOGY AND CLINICAL MANIFESTATIONS

When indelible pencil-lead is introduced into the body tissues it rapidly breaks up as the binder dissolves, leaving free particles of graphite and methyl violet; the latter, being readily soluble, diffuses into surrounding tissues. The basic dye then combines with the nucleic acid of the cells, killing them and resulting in an aseptic necrosis. This process continues peripherally until most of the dye is 'fixed' and its dilution is such that it is no longer toxic. It is only when this point is reached that the normal body defences can come into play, Erdheim (1914) describing a leucocytic barrier surrounding the lesion. Eventually the whole necrotic area will, if superficial, separate as a slough or, if deep, slowly discharge through a sinus. Finally, healing occurs by granulation and its concomitant fibrosis often leads to poor functional recovery of the part.

From case reports it would appear that these injuries usually bear stigmata that render their recognition easy. If the amount of 'lead' is small and implantation superficial, all that may result is a violet 'tattoo'. It was in such a tattoo that

Sharlet (1938) observed the development of a melanoma.

A recent lesion is usually characterized by blue staining of the puncture site and its exudate, and initially it would seem that these wounds tend to be more hæmorrhagic than a simple puncture.

The older lesions usually present in three main forms: a necrotic indolent ulcer, a proliferating granuloma, or a chronic sinus. All of these may show some degree of discoloration due to the dye. Pain and œdema are often marked, but anæsthesia has been recorded, presumably due to the involvement of sensory nerve-endings. It is of interest that these wounds are resistant to secondary bacterial infection, the methyl violet acting as a bacteriostatic agent.

The rarest form of this injury is the closed one. It is not an unusual story, in the form presenting as a sinus, for it to heal over for a brief period and later to break down again, but the closed type *per se* is rare.

All authorities are in agreement that the only form of treatment is a thorough excision of all involved tissue. Picking out the lead with forceps, simple incision, or débridement by curetting are all equally futile.

CASE REPORT

The patient, a young shorthand typist, was seen in the Hand Clinic of this Hospital giving a history of driving a pencil into her left hand 1 week previously. The puncture wound bled profusely and stung severely. Twelve hours later the wound was red and swollen and throbbed, but these signs had subsided by the third day and since then her only complaint was of stiffness in her index and middle fingers. On examination, she had a soundly healed unstained puncture wound, midway along the thenar skin crease. The puncture site was slightly tender to firm pressure, and there was no lymphangitis or lymphadenopathy. Flexion of all joints of the index and middle fingers was limited, this being most marked in the metacarpophalangeal joints, and an X ray did not reveal any radio-opaque foreign body. At this time she was carefully questioned as to the possibility of the pencil being an indelible one and she was most emphatic that it was not. This, taken with the paucity of physical signs, led to a diagnosis of a resolving cellulitis due to a simple puncture-wound being made, and it was treated conservatively by rest in a splint. A week later the picture had changed. She was now complaining of pins and needles in the two fingers, the tips of which felt numb and it was found there was partial anæsthesia in the distribution of the branch of the median nerve to these fingers. It was now thought that the signs could be explained by a piece of pencil lead lying adjacent to or in the nerve and it was decided to explore the nerve under a general anæsthetic.

On admission to hospital the degree of anæsthesia had increased, sensitivity to light touch had been lost, and that

FIG. 241.—Graphite particles lying in the tissues superficial to the palmar fascia.

FIG. 242.—Violet discoloration showing through palmar fascia which was the first indication of the true nature of this injury.

FIG. 243.—Condition at the end of the operation. Radially the affected branch of the median nerve is shown, the motor branch lying immediately proximal to the stained segment. The sublimis tendon to the middle finger lies medially, the more deeply stained surface of it having been pared away. The other sublimis and the profunda tendons presented a similar picture.

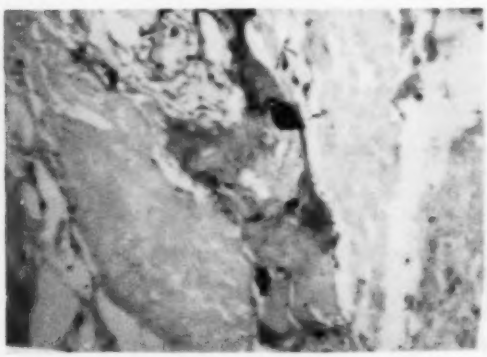

FIG. 244.—Photomicrograph showing the aseptic necrosis. The dark particles are deposits of graphite. (× 270.)

to pin prick was very impaired. For the first time there was a suspicion of a swelling beneath the puncture site and the area was painful when pressed. There were no signs of any systemic upset, pulse, temperature, white-cell count, and sedimentation rate all being normal.

Operative Findings.—In a bloodless field the area was explored via an incision in the line of the thenar skin crease. In the subcutaneous fat there was a collection of black granules thought to be graphite (*Fig.* 241). This was mobilized as a block and excised. This exposed the palmar fascia which was stained a pale violet over an area of 1 sq. cm. (*Fig.* 242). It was only then that the true nature of this injury was recognized. After excision of the involved fascia there appeared to be a dark violet necrotic tumour involving all the structures of the thenar side of the mid-palmar space extending down to the interossei. The first recognizable structure found was the superficial palmar arch which was thrombosed and was resected. The median nerve was then seen to be intact until just distal to the motor branch to the thenar muscles and the sensory branch to the first cleft. Beyond this the branch to the second cleft was deeply impregnated with dye for a distance of 1 cm. As the necrotic material was picked out it was found to consist largely of paratenon of the flexor tendons of the index and middle fingers, the medial part of the ulnar bursa, and the heads of the first two lumbricals. When exposed it was found that the opposing surfaces of the flexor tendons involved were also stained deeply. These areas were gently pared, removing most of the contaminated tissue.

The end-result was as seen in *Fig.* 243, the only dubious tissue not excised being the affected area of the median nerve. If a resection and end-to-end suture had been technically easy it would have been done; however, this would have necessitated a graft, and bearing in mind that clinically there was still some conduction through the affected segment a conservative policy was adopted. The wound was closed about a silkworm gut drain.

Histological Examination of material removed at operation showed an unusual necrosis of fibrous tissue with a chronic inflammatory reaction. Scattered areas of nuclear debris could be seen, and patches of black material presumed to be graphite (*Fig.* 244).

Post-operative Progress.—It was feared that despite almost complete removal of affected tissue fibrosis would ensue and the flexor tendons become adherent. In addition, it was uncertain how the involved segment of nerve would behave, particularly as to whether the contained dye would continue to give trouble. In an attempt to minimize these sequelæ it was thought that a cortico-steroid might help and the patient was placed on oral prednisone 10 mg. daily and active physiotherapy.

The wound healed by first intention. Following operation there was little improvement during the first month but when seen at 2 months the anæsthesia had lessened and the range of flexion of the affected fingers had improved considerably. Three months post-operatively it was noticed that one area of the scar seemed slightly hyperplastic and a skin biopsy was taken to see if the changes described by Mason and Allen (1941) were present but no evidence of epithelial hyperplasia could be found.

The last time this patient was examined was 6 months post-operatively. Sensation in the fingers was almost intact, there being only a slight deficit in light touch, and the only complaint the patient made was that this sensory loss made fine movements such as picking up a pin difficult. Flexion of the interphalangeal joints was full, but that of the metacarpophalangeal joint of the index finger was restricted, being 10° less than normal. The result of these defects was that the patient had to give up her job as a shorthand typist and engage in a less skilled and less lucrative employment, and it was two and a half months before she was able to resume work at all.

DISCUSSION

This case raises several points of interest, especially to the surgeon working in a casualty department or a hand clinic.

First, there is the question whether a puncture wound could be due to an indelible pencil: in this case it was considered and rejected. The patient, an intelligent girl, was emphatic that it could not be so and the accepted stigmata of indelible pencil injuries were not present. Thus the injury, already 1 week old when first seen, was not explored for a further 2 weeks. As to why this lesion was a silent one is difficult to explain, but presumably the strong palmar aponeurosis tended to confine it and prevent any superficial manifestations. However, the net result to this patient was a period of unemployment and the abandoning of her chosen occupation. It can be argued that the probability of meeting such a lesion is small these days especially since the advent of the ball-point pen, but this experience would suggest the wisdom of exploring immediately any pencil puncture wound unless the offending pencil can be produced for inspection.

Secondly, in dealing with such a lesion there is the question whether any dye-stained tissue at all should be left in situ. Perusal of the literature does not encourage one to do so. The question arose in this case over the involved nerve. It appeared deeply stained and its excision desirable, but, on the other hand, clinically some conduction was occurring along it and this, taken in conjunction with the smallness of the stained segment, led to a chance being taken, and it was left undisturbed. In view of the report by Hosford and Smith (1952) of the use of fluorescein it might be worth while if faced with a structure of which preservation is desirable, its non-viability not evidently manifest and yet deeply stained, to try to lyse out some of the contained dyestuff with fluorescein before closing the wound, or even to leave an indwelling polythene catheter in situ and irrigate the area with fluorescein for several days following exploration.

A third point of interest was the relative resistance of nervous tissue to the effects of the methyl violet. The affected segment of nerve was running through a completely necrotic area, the artery immediately superficial to it being dead and thrombosed, yet the nerve was still capable of some conduction and eventually recovered almost completely. Possibly the myelin sheath affords some protection from the penetration of the dye to the axon, but whatever the explanation it would seem from this experience that one can be more optimistic about nerves than other tissues and even if contaminated they can be left in situ with a reasonable hope of functional recovery.

Finally, there is the question whether the use of prednisone influenced the outcome of this case. It would appear that the final disability ensuing was much less than could have been reasonably anticipated from the operative findings: this may be accepted by the reader as some small evidence that the prednisone was beneficial in reducing fibrosis and adherence of the pared tendons.

SUMMARY

1. A young woman developed digital neuritis 2 weeks after a penetrating injury with a pencil,

which, contrary to her statement, was an indelible pencil.

2. Because of the serious effects of indelible pencil lead all pencil injuries should be explored unless it can be proved that the pencil is not indelible.

3. Prednisone post-operatively appeared to be beneficial.

Acknowledgements.—I am indebted to Professor R. S. Pilcher for his encouragement and advice in preparing this article, and to Mr. D. Ellis for the operative photographs.

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THE AETIOLOGY AND TREATMENT OF PILONIDAL SINUS

A REVIEW OF 163 PATIENTS

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IN 1854 Dr. J. Mason Warren reported 3 cases of "abscess of the nates connected with a fistulous opening over the coccyx containing hair". In 1877 he reported a further case, treated by incision and packing, in which he apparently had some difficulty in obtaining satisfactory healing, for it was six weeks before the patient was finally discharged well. Three years later Hodges (1880) suggested the term 'pilonidal sinus' for this condition.

This paper is a review of 163 patients suffering from this condition who have been admitted during the last twenty years to a large provincial teaching hospital. The results of the investigations suggest that some 105 years after Warren's original observation, difficulty is still encountered in obtaining satisfactory healing and we have advanced little on his original method. This difficulty may be indicated by the following summary which is expanded later:—

The average amount of time lost from work as a result of a sinus is some 13 weeks, of which 7 weeks are spent recovering from an operation designed to cure the condition. Despite this care and long period of incapacity, 1 in 4 patients required further surgery.

In view of this high failure rate, which is equivalent to that obtained in many other published series, methods of treatment were examined further to determine, if possible, any reasons for failure or guides to success. Since treatment is bound to be influenced by the pathogenesis of the condition, its natural history will first be reviewed in the light of details obtained from the present series.

PATHOLOGY AND INCIDENCE

Pilonidal sinus occurs most commonly in the upper part of the natal cleft, the primary sinus usually passing in a cephalad direction from the cleft. Further sinuses may pass laterally, these multiple sinuses occurring most commonly where the primary sinus has been grossly infected. Kooistra (1942) reported that lateral sinuses occurred twice as commonly on the left side as on the right, an observation not confirmed by the present series where abscesses and lateral sinuses occurred in approximately equal numbers in the two sides. The condition is not, however, entirely limited to this site. Jackson (1854) quoted a case in which a lock or pellet of hair was found in a specimen taken from near the umbilicus, and Hodges (1880) reported that the removal of hairs from beneath the skin of the feet was a practice repeatedly performed by 'pedicures'. Patey and Scarff (1946) reported the occurrence of typical pilonidal sinuses in the interdigital cleft of a barber's hand, and since that time similar sinuses have been reported in the axilla (Aird, 1952), perineum ('anterior perineal sinus'—Smith, 1948), and in a mid-thigh amputation stump (Shoesmith, 1953). In the present series, out of a total of 163 patients, 4 sinuses in uncommon sites were recorded—axilla, front of chest, umbilicus and the interdigital cleft of a barber's hand (*Fig.* 245). Since completion of the series a further anterior perineal sinus has been treated.

Table I.—AGE INCIDENCE IN YEARS

		Female	*Male*
Average age of onset	23·5	21·7	24·9
Average age of entering hospital	26·7	24·4	28·5

The figures of age incidence shown in *Table I* are comparable with the figures quoted by Kooistra (1942) of onset at 21¼ years and admittance at 25 years. The youngest in the series to require

treatment was 1 year 4 months, and the oldest 68 years. Sex incidence was: female 43 per cent (70 cases); male 57 per cent (93 cases).

Patients with sinuses present for treatment because of recurrent boils, pain, or chronic discharge. The incidence of the condition falls off rapidly after the third decade, and Klass (1956) assumes that this implies a natural course of the disease towards a

FIG. 245.—Pilonidal sinus in a barber's hand.

spontaneous cure between the ages of 25–30 years. A characteristic example of this is seen in the following case:—

W. W., a lady of 27 years, presented with a 4-year history of discharge and recurrent abscesses, and was admitted to the Radcliffe Infirmary under the care of Mr. D. C. Corry. A radical attempt at cure by wide excision was performed in 1942, but within 2 weeks the discharge had recurred and persisted, so that the following year a further wide excision was found to be necessary. Despite this she developed a true recurrence of the sinus with hairs present, and no further surgery was attempted. Symptoms persisted for the next 4 years and then ceased, and she has had no further trouble for the last 12 years.

However, as is well known, spontaneous cure is by no means universal, and on rare occasions the condition may progress to frank malignancy (Kleckner, 1936; Goldman and Kalow, 1940). Doubt has been cast as to whether true malignancy ever occurred in a sinus. For this reason it seems pertinent to quote the following case:—

W. B., aged 63 years, presented in 1950 with recurrent abscesses over the sacrum for the previous 20 years, and was admitted to the Radcliffe Infirmary under the care of Mr. D. C. Corry. A typical pilonidal sinus was widely excised. The histological report on this, which Dr. H. Cowdell has recently been kind enough to review, showed evidence of a typical squamous-celled carcinoma—a photomicrograph of the relevant part of the sinus is shown in *Fig.* 246. The patient subsequently developed a recurrent sinus which was again widely excised in 1951, the histology showing no evidence of recurrence of malignancy. The wound, which was sutured, has remained soundly healed since then.

AETIOLOGY

This has been perhaps the principal point of issue in the discussion of this condition. Certain factors have been cited as having a bearing on the aetiology of the condition—notably chronic irritation,

FIG. 246.—Squamous-celled carcinoma in long-standing pilonidal sinus. H. and E. (× 47.)

sepsis and trauma, and the occurrence of the lesion typically in hirsute patients. Trauma was shown to be a common antecedent during the war when the familiar term 'jeep-seat' was coined. In civil practice a definite history of injury is uncommon, and was obtained in only 23 out of 163 patients, but it was frequently ascribed by patients to an occupation resulting in repeated minor injuries, as for example in a college oarsman, and in a dance-band drummer. The latter patient volunteered the information that it was generally regarded as a common condition 'in the profession', and it might perhaps be registered as a rather exclusive occupational hazard—'drummers' seat'.

It is not the purpose of this paper to put forward new evidence on the problem of aetiology, but some opinion is of importance since it must influence views on treatment. Thus Kooistra (1942) stated, "it is obvious from the pathology that any form of treatment to be successful must completely eradicate the cyst and sinuses, and all granulation tissue". On the other hand, Patey and Scarff (1946) after presenting evidence to suggest that in the great majority of cases the lesion is primarily infective, with secondary entrance of hair and debris, stated that "emphasis should be shifted from wide eradication by excision to the management of a chronic infective lesion in a fold".

The various theories of aetiology have been reviewed by Hopping (1954). They may be summarized here:—

1. Ingrowth of hair from a single follicle—an acquired condition (Warren, 1877).

2. Congenital imperfect union of the two halves of the body, involving integument only, with subsequent invasion by hairs and dirt, and consequent sepsis (Hodges, 1880).

3. Hereditary cicatrix of a spina bifida by which the human tail was lost (Tait, 1878).

4. Coccygeal medullary vestiges of a neural canal located in the subcutaneous tissues over the sacrum and coccyx (Tourneaux and Herrmann, 1887).

5. The vestiges described above, however, consist of differentiated cells which cannot give rise to true skin, and Streeter (1924) believed that pilonidal disease was due to local downgrowth of epithelium originating from true skin.

6. The vestiges might be related to the preen gland in birds (Stone, 1931).

7. There is a firm fibrous connexion between skin and bone in the sacrococcygeal region during early embryonic life, and neurodermal tissue grows in from the sides, elevating the skin around the attached spots forming clefts and cysts (Mallory, 1892; Oehlecher, 1926). Gage (1935) described four grades of sinuses:—

a. Sacrococcygeal dimple—'dimple sinus'.

b. True pilonidal sinus.

c. A sinus extending down between the sacrum and coccyx and entering the sacral canal, even entering the dura.

d. A sinus communicating with the central canal of the spinal cord.

8. Fox (1935) suggested the condition was a skin appendage developing to maturity at puberty.

9. A vestigial sex gland (Kallet, 1936).

10. A foreign body granuloma produced as a result of penetration of the skin by hair—primarily an infective lesion with secondary entrance of hair and debris introducing both infection and epithelium. Patey and Scarff (1948), developing this theory of acquired origin, suggested that the hair is "sucked into a sinus, which is primarily infective". Brearley (1955) added further to this by postulating a sinus commencing by puncture of the skin by hair—the puncture sinus. This is followed by the hair being sucked in further by negative pressure developed deep to the sinus which is produced by lateral movement of the buttocks elevating the skin over its attachment to the coccyx. Brearley provided experimental evidence that such a negative pressure is in fact developed.

Patey and Scarff (1955) also pointed out that the arrangement of scales in a hair is such that friction along the hair, from its periphery to its root, will produce movement toward the root, but not in the reverse direction. This would help it first to puncture the intact skin, and secondly impede its leaving the sinus.

In favour of the suggestion of Brearley that suction force is of importance the following case is of interest:—

H. T. H., aged 24 years, presented in 1955 with a 4-year history of a lump in the front of the chest, with a recent exacerbation of discharge. This exacerbation had followed a week-end spent clipping his hedges. He was admitted to the Radcliffe Infirmary under the care of Mr. D. Abernethy, and the lump was excised. Histology showed this to be a typical pilonidal sinus. He made an uninterrupted recovery, and has had no recurrence.

The vigorous lateral movement of the arms required in clipping a hedge could produce in a man of good muscular development a similar negative pressure deep to the pre-sternal skin, and so provide conditions for the development of a suction sinus. It should also be pointed out that similar conditions exist in other less common sites of pilonidal sinuses, namely, in the axilla and interdigital clefts.

A further point against the condition being due to a congenital lesion is its relative rarity in young infants. Undoubtedly the 'dimple sinus' does exist, but this would seem to have a different natural history from that of a typical pilonidal sinus, as is illustrated in the following two cases:—

J. H., aged 1 year 11 months, presented in 1946 with an infected sinus present for 18 months and was admitted to the Radcliffe Infirmary under the care of Mr. D. C. Corry. Operation was contra-indicated by the presence of an associated dermatitis. He was treated with penicillin, and made a satisfactory recovery. He has had no further trouble in the succeeding 13 years.

A. S., aged 16 months, presented also in 1946 under Mr. Corry's care, was treated similarly, and also had no further trouble. Since this course is so different from that of the adult lesion, it is suggested that the lesion is of a different nature.

There are thus two main theories of origin which imply differing emphasis in treatment thus:—

1. If it is a congenital sinus arising from some embryonic vestige, surgical treatment would be most effective if it completely eradicated the lesion. Wide excision should then give the best prospect of cure.

2. If it is an acquired lesion—"a chronic infective focus at the bottom of a fold"—treatment is aimed at securing satisfactory healing of this, and excision is not a primary necessity.

TREATMENT

In the patients included in this review, treated at the Radcliffe Infirmary and Churchill Hospital, Oxford, there have been four main methods of surgical treatment:—

Table II.—Recurrence Rate following all Forms of Surgical Treatment

	No. of Surgical Interventions	No. of Cases followed for over 1 year	Recurrences Number	Per cent
Incision of acutely inflamed abscess	41	40	37	92·5
Wide excision and primary suture	126	110	42	38·2
Wide excision and healing by granulation	36	29	6	20·7
Incision and healing by granulation	15	13	1	7·7

1. Incision and drainage of an acutely inflamed pilonidal abscess.

2. Wide excision of the whole lesion and primary suture.

3. Wide excision of the whole lesion leaving the wound open to allow 'healing from the bottom' by granulation.

4. Simple incision and laying open of the sinus to allow healing, without wide excision.

The 'recurrence rate' of these measures has been simply assessed by two criteria:—

a. Was further surgery necessary?

b. Was the patient relieved of his or her symptoms?

The results are tabulated in *Table II*. The recurrence rate was assessed by comparing the number of recurrences with the number of patients symptom-free for at least one year after surgery. Patients followed for less than one year, even if free of symptoms, are not included in calculating this figure.

1. Incision of an Acutely Inflamed Pilonidal Abscess.—Forty-one patients presented for surgery with an acutely inflamed abscess, which was incised and drained by Hilton's method. Thirty-five were considered within 1 year to require further surgery, while 2 patients remained symptom-free for over 2 years before developing recurrent abscesses requiring further surgery. Four patients had no further trouble. One has been followed for only 1 year, the other three for 3–11 years. The recurrence rate for this group of patients (37 out of 40) is 92·5 per cent.

Table III.—Periods of Follow-up of Symptom-free Patients Treated by Wide Excision with Primary Suture

Less than 1 yr.	1	2	3	4	5	6	7	8	9	10	more than 10 yr.
16	8	7	5	3	7	10	4	6	3	3	12

Table IV.—Interval between Operations and Recurrence in Patients Treated by Wide Excision with Primary Suture

1 mth.	6 mth.	1 yr.	2 yr.	11 yr.
14	19	3	5	1

Table V.—Periods of Follow-up of Symptom-free Patients Treated by Wide Excision and Healing by Granulation

Less than 1 yr.	1	2	3	4	5	6	8 yr.
7	4	6	5	1	4	2	1

Table VI.—Periods of Follow-up of Symptom-free Patients Treated by Incision and Healing by Granulation

Less than 1 yr.	1	2	3	5	10 yr.
1	6	3	2	1	1

2. Wide Excision with Primary Suture.—On 126 occasions patients were treated by wide excision and primary suture. Eighty-four patients obtained satisfactory results, as shown in *Table III*. Excluding the 16 patients followed so far for less than 1 year, 68 out of 110 patients were 'cured'. In 42 cases further surgery was required, or offered and refused, or else the patients had further disabling symptoms.

Table IV shows the time interval between operation and the onset of further symptoms ultimately leading to further surgery.

It will be seen that the majority of recurrences (36 out of 42, or 85·7 per cent) occurred within the first year. The recurrence rate for this group of patients (42 out of 110) is 38·2 per cent. A further analysis of the factors leading to recurrence will be made later.

3. Wide Excision and Healing by Granulation.—This was performed on 36 occasions. Thirty patients became symptom-free with the periods of follow-up shown in *Table V*. There were 6 recurrences, 4 of which occurred within 6 months, while 2 patients remained symptom-free for over 5 years before the onset of further trouble. Twenty-three patients have obtained satisfactory results with a follow-up over 1 year, while 6 were failures. The recurrence rate for this group is therefore 20·7 per cent.

4. Incision of Sinus Track with Healing by Granulation.—Fifteen patients are available for study. Of these one required further surgery, 6 months later. A second patient required division of a bridge of skin which had formed as a result of too rapid healing of the granulating area. This was a minor procedure, and the patient is not therefore classed as a 'recurrence'.

The periods of follow-up for the patients who had no further trouble are shown in *Table VI*.

Thirteen patients are available for follow-up of more than 1 year symptom-free, while there was 1 recurrence. The recurrence rate of this group is therefore 7·7 per cent.

FACTORS RESPONSIBLE FOR RECURRENCE

Consideration was then given to the factors which may have a bearing on the incidence of recurrence. The following factors were studied: (1) The presence or absence of infection at the time of surgery; (2) The routine use of chemotherapy; (3) Completeness of excision as measured by the use of methylene blue as a guide to the extent of the lesion; (4) Following excision and primary suture, whether or not primary healing was obtained; (5) Whether any form of drainage was used at the time of excision and primary suture; (6) The use of X-ray depilation post-operatively. These will be considered in turn.

1 and 2.—The effect of infection at the time of surgery, and the use of chemotherapy. Cases were classed as infected if there was any acute inflammatory process clinically obvious at the time of surgery,

Table VII.—Presence of Infection and Use of Chemotherapy: their Influence on Recurrence Rate

	Wide Excision and Primary Suture			Excision and Laying Open			Incision and Laying Open		
	No.	Recurrence	Per cent	No.	Recurrence	Per cent	No.	Recurrence	Per cent
Infected—chemotherapy	25	9	36·0	2	1	50·0	6	0	0
Infected—no chemotherapy	22	8	36·4	15	2	1·3	1	1	100
Non-infected—chemotherapy	29	11	37·9	4	1	25·0	0	0	0
Non-infected—no chemotherapy	28	6	21·4	11	0	0	8	0	0

or if there was a chronic purulent discharge. The results of 151 surgical interventions in which records supply the necessary information are classified in *Table VII*.

In the smaller series, the exact figures of recurrences are not of significance, though it is apparent in the relatively larger series treated by wide excision and primary suture that chemotherapy made no difference to the incidence of recurrence. This conclusion was also reached by Pecora and Cooper (1955) after a similar investigation. The apparent discrepancy in the results achieved in non-infected cases where chemotherapy was used (a recurrence rate of 37·9 per cent) shows the disadvantage of adopting a rigid dividing line between infected and non-infected cases. Though neither an acute inflammatory process nor a chronic purulent discharge was present at the time of surgery, the surgeon concerned presumably advised the use of chemotherapy because he considered that some contamination may have occurred. It is apparent from the results that any contamination greatly increases the incidence of recurrence where primary suture is adopted.

Table VIII.—The Use of Methylene Blue; its Effect on Recurrence Rate

	No. of Patients	*Recurrences*	*Recurrence Rate*
Methylene blue used	60	21	35 per cent
Methylene blue not used	81	23	28·4 per cent

Table X.—Influence of Primary Healing and Infection on Recurrence Rate

		No.	Recurrence No.	Per cent
Primary healing obtained	Infected cases	23	9	39·1
	Non-infected cases	32	4	12·5
Primary healing not obtained	Infected cases	20	7	35·0
	Non-infected cases	23	11	47·0

Table XI.—The Use of a Drain and its Influence on Primary Healing and on Recurrence Rate

	No. of Patients	Primary Healing Obtained		Recurrences	Per cent
		No.	Per cent		
Excision and primary suture with drain	30	18	60·0	10	33·3
Excision and primary suture without drain	61	46	76·7	12	20·0

Note: These figures include some cases in which there was no recurrence although primary healing was not obtained.

3.—Effect of complete or incomplete excision. Evidence has already been presented showing that it is not necessary to excise the lesions entirely in order to obtain a cure—better results follow incision than excision. Also, in a few cases where the pathologist examined the periphery of the excised specimen and reported the lesion as being completely excised, recurrences still occurred. Methylene blue is used by some surgeons to show the extent of the lesion by its injection into the sinus prior to excision. Records are available of the use or omission of methylene blue in 141 operations, and *Table VIII* shows that its use made little difference to the recurrence rate.

Table IX.—Influence of Primary Healing on Recurrence Rate

	No. of Patients	*Recurrences*	*Recurrence Rate*
Primary healing obtained	55	13	23·5 per cent
Primary healing not obtained	43	18	41·9 per cent

4.—Where a sinus has been excised with primary suture, and primary healing obtained, it is usual to assume that a cure has been achieved. This assumption was re-examined in the light of the present series. Records were available of whether or not primary healing was obtained in 98 cases with adequate follow-up—over 1 year (*Table IX*). These showed that, as might be expected, if the wound breaks down, the chance of recurrence is increased.

The series was further examined in an attempt to find some indication of selection for this particular surgical procedure. These results are also as might have been expected, i.e., the best results of primary suture are obtained in operations on apparently uninfected lesions. If the case is infected, however, whether or not primary healing is obtained, the results do not compare with laying open the area and leaving it to heal by granulation either with or without wide excision. In addition, it was quite clear that if primary suture is to be employed, no effort must be spared to achieve primary healing. If the wound breaks down, a poor result can be expected, those in the present series being the worst of any group (*Table X*).

Table XII.—Recurrence Rate with and without Drainage, from Kooistra (1942)

	Recurrence
Primary suture with drainage	34 per cent
Primary suture without drainage	21 per cent

5.—The use of a drain after excision and primary suture to reduce hæmatoma formation carries the added risk of leaving a track liable to infection, and this may decrease the likelihood of achieving primary healing. The series was analysed with this factor in view. Records are available of 91 cases with follow-up of one year or more. The figures indicate that the routine adoption of drainage does not increase the chances of obtaining primary healing, and in the present series was followed by an increased recurrence rate. It is of interest that the figures compare closely with those found by Kooistra (1942) (*Tables XI* and *XII*).

6.—Since hairs in a sinus are probably one factor in ensuring its persistence and leading to its recurrence, X-ray depilation has been occasionally used after surgery. In the present series, though the

numbers involved are small, and probably of no statistical significance, they are of some interest (*Table XIII*).

It is now generally considered that the dose of X rays required for depilation is too variable, and the dangers of X rays too great to warrant their use in

Table XIII.—Use of X-ray Depilation

No. of patients	10
Recurrences	4

benign conditions. In view of the recurrence rate of 40 per cent found in this series despite depilation, it would not seem to have any place in treatment.

INCONVENIENCE AND COST

Since approximately equivalent results can be obtained either by excision and primary suture in the 'ideal case', or by incision and laying open in any other case, it may be questioned whether any advantage is to be gained by attempting primary suture in any case, even the 'ideal'. This prompted an analysis of the length of time spent in hospital, and subsequently off work before resuming normal employment, and all patients were circularized to find this latter figure. There were 115 replies to this questionnaire (*Table XIV*).

From this it will be seen that little advantage is to be gained by attempting primary closure even in

Table XIV.—Relation between the Method of Treatment used and the Length of Subsequent Disability—in Days

	Average Time in Hospital	Average Time off from Work following Surgery
Excision and primary closure	18·3	57
Excision and primary closure. Primary healing obtained	14·4	45·4
Excision and laying open	18	53
Incision and laying open	11·5	37

the 'ideal case'. It is startling to note that the cost to the Exchequer in terms of the cost of hospital beds and in sick pay for each patient is in the region of £60–100, depending on the type of treatment employed. This does not include the loss of normal earnings of the patient.

DISCUSSION

The principal object of this paper is to analyse the results obtained following attempted radical surgical cure of a pilonidal sinus by a large group of surgeons. The recurrence rates obtained in this series are approximately in accordance with those of other writers, though occasionally writers have claimed a lower rate. For example, in a review of the treatment of 4596 cases carried out by the American Proctological Society, the use of the 'open method' was followed by a 1·13 per cent recurrence rate (Kleckner, 1936). Unfortunately no follow-up time is given, and it is difficult, therefore, to compare the results with those of the present series.

As already mentioned, it is impossible not to form some opinion on the aetiology of the lesion, and it is important that this should be so, since practice depends on pathology. My own opinion from this study is that the vast majority of sinuses is acquired, and only a small minority is congenital. It is even possible that the congenital lesions are essentially of a different pathology from the acquired. The importance of this in practice is that total excision is not synonymous with cure, and is not essential.

Methylene blue has been used in an attempt to achieve total excision, and its use may be considered at this stage. It was found to make little difference to results in the present series (*Table VIII*). Opinions vary on this, and Whyte (1938) maintains that a false sense of security is obtained, as occluded channels do not show stain, normal tissue is stained, and wider excision than necessary may be performed. Kooistra (1942) summarizing the literature wrote that though the data appear inconclusive they favour the school recommending pre-operative injection, while McKinney (1936) quotes a case where the dye led round the edge of the sacrum and coccyx to the front of the sacrum where there was a tumour present, which would have been missed by the usual surgical procedure. However, methylene blue is of little value in the majority of cases, and there seems little to indicate its use as a routine procedure.

As regards the surgical procedure to be adopted, the present series leaves little doubt that incision and packing gives results at least as good as any other procedure, and better than most. In addition, the time required off work is appreciably shorter after simple incision and packing than after wide excision whether or not primary closure is attempted. This is partly because the area left by simple incision is smaller than the large raw areas left by block excision. In addition, the proximal part of the sinus is lined by skin, and islands of this are left on the surface of the healing area, to take part in the process of epithelialization. Gabriel (1949) points out the necessity for selecting cases for excision and primary suture with great care, but there would seem to be little advantage gained from this procedure, even in the so-called 'ideal case'. Even if primary healing is obtained the cure rate is no higher, and the hospitalization period is longer than that obtained by incision. Primary skin healing provides no guarantee against subsequent recurrence; it may merely provide a temporary covering for a persistent lesion. If the attempt at primary healing fails, the recurrence rate is at least five times that achieved by simple incision. It is interesting to compare this conclusion with that expressed by the American Proctological Society (Kleckner, 1936) in which a review of answers to a questionnaire sent to all members of the society "showed a definite trend away from massive block excision and toward limited excision with partial closure or marsupialization".

If simple incision and packing is to be adopted, could it not also be applied to the treatment of acutely infected lesions? Dwight and Maloy (1953) claimed a 93 per cent cure rate with abscesses, giving a follow-up of at least one year. In the present series, in which such abscesses were treated simply by a small incision and drainage, this produced a recurrence rate of 93 per cent. The prime disadvantage of incision

of the whole sinus and all its tracks is that it entails a much longer period in hospital and subsequently off work, and the average patient is not prepared for this when presenting 'as an emergency'. However, where it is economically possible, it should certainly be advised.

If a more simple technical procedure is to be adopted then adequate supervision of after-care becomes of prime importance. While depilation by X ray has been shown to have no place in treatment, no one who has followed the healing of these wounds can fail to be impressed by the rapidity with which hairs at the side of the healing area soon contaminate the granulation tissue, and this area should be shaved regularly until such time as healing is complete. In addition, bridges of skin rapidly form across the healing area when healing is nearly complete, and overlie potential recurrent sinuses. It is at this time that supervision is most important and epithelial bridges should be broken down, thus ensuring slow healing by the spread of epithelium from the edges over flat granulation tissue in the bottom of the fold without bridge or tunnel formation. Subsequent depilation can be achieved by the regular use of fine glass-paper, and in hirsute patients this is worthy of a trial.

CONCLUSION

1. Most pilonidal sinuses are of acquired origin, though, rarely, congenital lesions do occur.

2. Massive block excision is not necessary. The whole lesion need not be removed, and the use of methylene blue is not indicated.

3. No advantage is gained by excision and primary suture, and its use is fraught with danger.

4. Primary skin healing does not mean cure. Recurrences may occur at any time, even ten years after apparently satisfactory healing has been obtained.

5. Incision and packing is strongly recommended as the primary technique in operative treatment.

6. If the circumstances of the patient permit, the same technique should be applied to acutely inflamed lesions.

7. Adequate supervision of after-care is the most important consideration in treatment.

8. Depilation by X ray carried no advantage; its use may be dangerous, and it should be condemned. Depilation can, however, be achieved by the use of fine glass-paper, and this should be recommended for hirsute patients.

Acknowledgements.—I wish to acknowledge with gratitude the help of the surgeons at the Radcliffe Infirmary and Churchill Hospital, Oxford, in allowing me to make use of their case notes, and to follow up their patients; and particularly Mr. A. S. Till and Mr. G. E. Moloney for allowing me to operate on some of their cases. I would also like to acknowledge Dr. Cowdell's help with the histology of the patient who developed a carcinoma. Finally, I would like to record my indebtedness to Mr. G. E. Moloney for his constant help and encouragement in the preparation of this paper.

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EXPERIMENTAL SURGERY

CAROTID ARTERY RECONSTRUCTION WITH THE AID OF PERFUSION

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THE invasion of the carotid arterial system by malignant glands in the neck has been regarded as a contra-indication to surgery, the reason being that excision with interruption of the cerebral circulation would be required and as a result there would be a high incidence of cerebral damage. There is considerable variation in the degree of collateral circulation to a cerebral hemisphere and it is upon this adventitious blood-supply that cerebral function depends in the post-operative period. Although it is possible in some cases to ligate the common carotid (Rogers, 1947) or even the internal carotid artery (Schorstein, 1940) with no neurological sequelæ, this is not so in all cases, and there is at present no satisfactory way of selecting those cases which will develop and maintain a suitable collateral circulation.

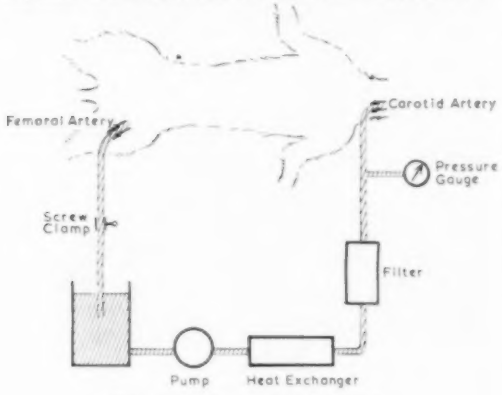

FIG. 247.—Extracorporeal circuit used.

We recently had such a patient develop hemiplegia after excision of a carotid body tumour. Expecting this tumour to be closely adherent to the vessels, the patient was cooled by surface ice packs until an œsophageal temperature of 30° C. was obtained; excision or ligation of the carotid vessels was not required, but it was necessary to cross-clamp the common carotid artery on six occasions though at no time for longer than 5 min. Post-operatively, a hemiplegia was present.

Two ways were considered in which the cerebral circulation might be maintained while the carotid vessels were excised and reconstructed. The first was the use of a temporary conduit from the proximal part of the common carotid to the distal part of the internal carotid artery near the base of the skull. This entails awkward technical disadvantages particularly if the growth is extensive, because a clear area is necessary both above and below it for the conduit to be attached. The second method, which was that chosen, was the use of an external circuit by which blood was withdrawn from the femoral artery and pumped back into the distal part of the internal carotid artery. By this means an adequate cerebral circulation was maintained while tumour and carotid vessel were excised and an arterial reconstruction performed.

METHOD

The preliminary routine was developed in the laboratory. A cannula was inserted in the femoral artery of a dog, and from it a plastic tube fitted with an adjustable screw-clamp led to a reservoir. The circuit was completed by an arterial pump (Mono) (Hall, James, Lucas, and Waterston, 1959), a heat exchanger (Brown, Smith, and Emmons, 1958), and a filter. A plastic tube ran from the filter to a cannula which could be inserted into the carotid artery of the dog. In this latter line was a straight metal connector with a side tap connected to a pressure gauge. This circuit is shown in *Fig.* 247.

FIG. 248. *J*-shaped arterial cannula.

In the dog a branch of the external carotid artery was dissected out and the arterial pressure in it recorded; when perfusion was started we maintained this arterial pressure at a normal level by suitably increasing the flow. The volume of blood taken out of the femoral artery was controlled by the screw-clamp, and by keeping the reservoir at a constant level, having previously primed the circuit with blood, we were able to maintain the dog's blood-volume at a normal level.

It was found that cerebral perfusion could be started in under 2 min. from the cross-clamping of the carotid artery. The time taken to start normal cerebral blood-flow after the perfusion had been

ended was longer owing to the need to repair the arteriotomy, and on one occasion this took 5 min. It was for this reason that we introduced a heat exchanger into the circuit, enabling us to produce hypothermia of one cerebral hemisphere and thus allow ourselves up to 10 min. in which to re-establish the normal circulation after the end of perfusion.

Dogs were heparinized with a dosage of 2·5 mg. per kilo before the by-pass was started. After surgery this heparin was neutralized with polybrene 3·75 mg. per kilo (Rothnie and Kinmonth, 1960).

In the human we estimated the arterial pressure in the carotid system by measuring the pressure in the perfusion line just proximal to the patient. The internal carotid artery was found to be about 5 mm. in diameter and accommodated a large cannula; there was no pressure drop at this point at the small flows we used, so that the above reading was considered reliable.

CASE REPORTS

Case 1.—Mrs. S., aged 64 years, developed a secondary malignant mass in the left side of the neck eleven months after treatment for an ulcerating squamous-celled carcinoma of the pyriform fossa. This mass measured 2 in. by 1 in. and lay in the region of the carotid bifurcation; it was attached to the deep structures. The internal carotid artery was felt pulsating above it.

At surgery a routine block dissection of the neck was carried out and it was found that the tumour had involved the common carotid artery. The common carotid artery was dissected out below the adherent mass and the external and internal carotid arteries were dissected out above. After cross-clamping the internal carotid an arteriotomy was made in it in its highest part and the cannula, previously primed with blood, was inserted and tied in position; perfusion was immediately started. The arterial cannula used was a J-shaped subclavian artery perfusion cannula, internal diameter 3 mm., made by the Genito-Urinary Manufacturing Co. This is shown in *Fig.* 248. A second clamp was then placed across the common carotid artery and excision of the gland and an adherent segment of the common carotid artery performed. End-to-end anastomosis of this artery was then carried out.

Blood was perfused at 215 c.c. per minute at a mean pressure of 70 mm. Hg. The perfusion lasted 20 min. in all; during the last 7 min. the temperature of the perfused blood was reduced to 30° C. in order to induce hypothermia of the left cerebral hemisphere, which would give protection from anoxic damage during arrest of the circulation through the internal carotid while the arteriotomy was repaired. In this case it took under 3 min. to repair the arteriotomy and establish normal circulation after the cessation of perfusion.

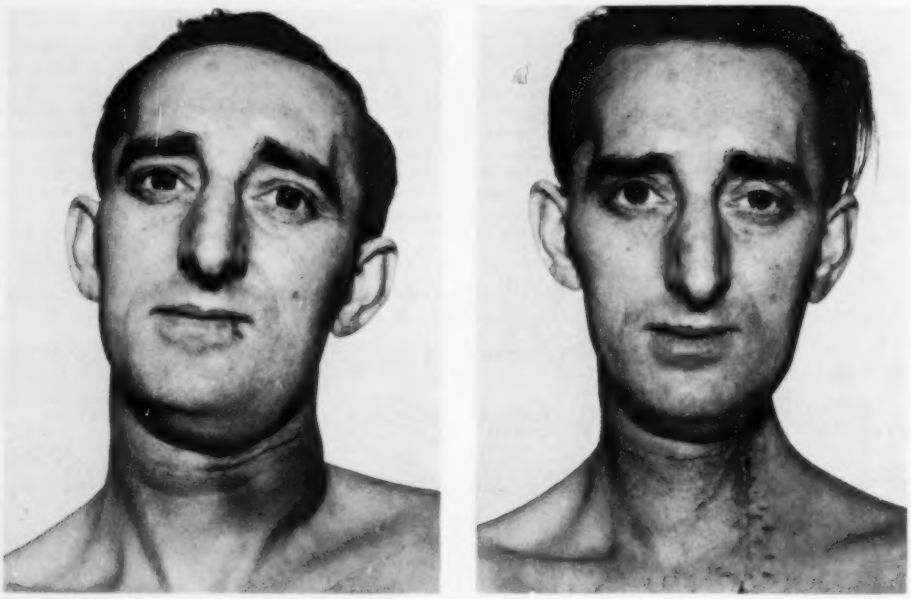

FIG. 249.—*Case* 2 before and after operation.

Post-operative recovery was perfectly satisfactory; there were no neurological sequelæ.

Case 2.—Mr. S., aged 33 years, developed a metastatic deposit in the left side of his neck one year after treatment for carcinoma of the larynx. A single mass, 3 in. by 2 in., was present lying under the sternomastoid, to which it was attached; it was also fixed to deep structures; arterial pulsation in the carotid system was palpable above the mass.

At surgery, a block dissection was carried out and the malignant mass found to be adherent to the carotid bifurcation and the vagus. After cross-clamping the common carotid artery the internal carotid artery was cannulated and perfusion commenced.

Resection of the block of tissue including the mass, the vagus nerve, and the carotid bifurcation was then performed. An arterial homograft 9 cm. in length was inserted to restore arterial continuity, the external carotid being ligated.

Blood was perfused at a flow of 205 c.c. per minute at a mean pressure of 100–120 mm. Hg. The perfusion lasted

3 min. in all; during the last 5 min. the temperature of the inflowing blood was reduced to 30° C. After the perfusion was terminated it took 5 min. to re-establish normal circulation.

Post-operative progress was satisfactory; there were no neurological sequelæ. Pre-operative and post-operative photographs are shown in *Fig.* 249.

DISCUSSION

The degree of cerebral damage resulting from ligation of either the common carotid artery or the internal carotid artery varies from case to case, depending on the collateral arterial blood-supply to the brain. This collateral circulation may be tested by exerting continued pressure on the carotid vessel and watching for either electro-encephalographic changes or subjective sensory changes in the patient. It may also be demonstrated radiologically by injecting radio-opaque material into one carotid while compressing the other. Though these methods may detect cases in which the collateral circulation is very inadequate, they are not completely reliable.

The earlier recorded series of carotid ligation included many cases with hæmorrhage from infected bleeding neoplasms and the mortality was high. Pilz (1868) collected 285 cases of ligation with a mortality of 52 per cent, and De Fourmestraux (1906) showed a mortality of 54 per cent. Sepsis, anæmia, and old age are considered factors in this high figure. As recently as 1939 Watson and Silverstone recorded 50 per cent hemiplegia resulting from common carotid ligation in cases of ulcerating neoplasms. Recently carotid ligation in the treatment of cerebral aneurysms has given more reliable figures, but these cases may already have prior cerebral damage and the neurological complications may be a result of the aneurysm itself rather than the ligation. Schorstein (1940) collected 49 cases of internal carotid ligation of which 8 died and 11 developed hemiplegia. Lambert Rogers (1947) found that 4 out of 6 cases with internal carotid ligation developed persistent neurological damage. Harris and Udvarhelyi (1957) found that 5 cases developed hemiplegia out of 22 with internal carotid ligation. All authors are agreed on there being some cases with persistent neurological damage.

With ligation of the common carotid artery the reports are more variable. Schorstein (1940) quotes 11 cases with ligation and no neurological damage, and Rogers (1947) 7 cases with no sequelæ. Harris and Udvarhelyi (1957), on the other hand, found 8 hemiparesis cases in 20 ligations and Shenkin, Pulakoff, and Finneson (1958) found 1 hemiparesis in 4 cases.

Perhaps the most reliable figures are those of Siegrist (1900) quoted by Homans (1920), who found among 113 ligations for pulsating exophthalmos an 8·5 per cent mortality, and Makins (1919), who found in 85 cases of gunshot wounds of the common carotid artery that 7 per cent died of cerebral complications.

Age is undoubtedly a factor in prognosis after carotid ligation, for the collateral circulation does not function so well in the elderly. Kocher in 1903 stated: "Ligature of the common carotid in an old man with arterial sclerosis is equivalent to the death sentence"; and Aird (1957) says that "except in young persons, and perhaps even in these, ligature of the common carotid artery is apt to be followed by hemiplegia".

The blood-flow to the brain has been estimated at 54 c.c. per 100 g. of tissue. The weight of the human brain is 1200 g. (Gray, 1947), thus the estimated blood-flow would be 648 c.c. per minute. This is divided between the two internal carotids and the basilar artery; as these three vessels are of equal size (Dorrance, 1934) it was thought that a flow of 200 c.c. per minute should be given and increased if the recorded arterial pressure in the carotid line was low. Our first perfusion was run at 215 c.c. per minute and gave a mean line pressure of 70 mm. Hg; the second was run at 205 c.c. per minute and gave a mean line pressure varying between 100 mm. and 120 mm. Hg.

The use of hypothermia in the last few minutes of perfusion enabled the arteriotomy in the internal carotid artery to be carefully repaired without pressure of time. The carotid arterial flow was kept at the same rate during this perfusion, but the temperature of the ingoing blood was reduced from 37° C. to 30° C. by means of the heat exchanger in the circuit.

SUMMARY

Excision of the carotid artery with reconstruction was carried out in 2 cases.

Blood-supply to the brain was maintained by an arterial line from the femoral artery to the carotid.

Blood cooling was carried out in the later stage of the operation to give the fullest protection from cerebral anoxia.

We wish to thank Mr. N. G. Rothnie and Dr. M. Steele for their help; also Miss S. Maughan, Miss D. Hulkes, and Mr. D. Rutt for their valuable technical assistance.

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SURGICAL AND PHYSIOLOGICAL OBSERVATIONS IN AN EXPERIMENTAL PRESSURE CHAMBER

By C. F. W. ILLINGWORTH, GEORGE SMITH, D. D. LAWSON, I. McA. LEDINGHAM, G. R. SHARP, AND J. C. GRIFFITHS, with technical assistance by C. I. HENDERSON

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THE effects on the solubility of oxygen in the blood and tissue fluids that can be produced by raising the ambient pressure have been understood for nearly a hundred years. Pressure vessels were in use at about the turn of the century for the treatment of respiratory affections, but they proved ineffective, perhaps because the pressure at which they operated was insufficient. More recently Churchill-Davidson, Sanger, and Thomlinson (1955) have made use of a pressure chamber as an aid to radiotherapy in the treatment of malignant disease. Their experience, and that of others since that time, has shown that within the pressure ranges used the procedure is devoid of risk. Still more recently Boerema (1961) has reported on the use of a pressure chamber for various surgical purposes.

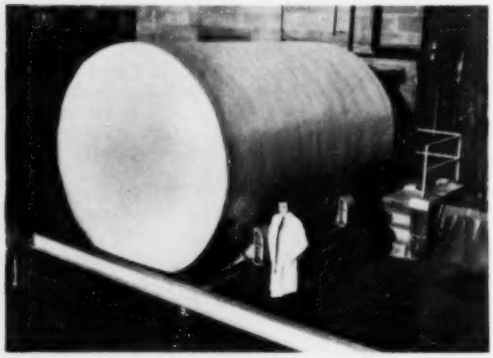

FIG. 250.—Exterior of pressure chamber.

Here in Glasgow work in this field began in 1956, using a makeshift chamber constructed from a hospital autoclave. The first step was to determine whether the administration of oxygen at high ambient pressure would mitigate the effect of sudden occlusion of the main blood-supply to a part. As a test procedure the method used was to ligate the circumflex branch of the left coronary artery of the dog, a procedure which is known in ordinary circumstances to carry an immediate mortality from ventricular fibrillation in 50 or 60 per cent of cases.

The success of these experiments (Smith and Lawson, 1958) was such as to justify further observations, both in animals and in clinical subjects. At this time support was gained from the Advisory Committee for Medical Research in Scotland, and on their recommendation a grant of £10,000 was obtained from the Department of Health for Scotland to construct a pressure chamber suitable for experimental work and also for clinical observations. The chamber was installed in June, 1960, and has since been in active use for a variety of purposes. We believe the time is now ripe for a preliminary report on the experience we have gained.

The Experimental Chamber.—The chamber, made of ⅜-in. welded steel plates, is of cylindrical shape, 18 ft. long by 11 ft. 6 in. in diameter. It comprises a main chamber 14 ft. in length and a small air lock. Its outer aspect is lagged to prevent heat loss. Its inner aspect is lined with acoustic insulation. The total weight is 12 tons (*Figs.* 250–252).

The pressure is raised by a motor compressor and an ingenious system of valves ensures a copious exchange of air. The temperature is under thermostatic control. Within the chamber the patient (or the experimental animal) breathes oxygen administered by a face-mask.

The chamber is designed to work at 2 atmospheres (absolute). This pressure was chosen because it is sufficient to provide a considerable increase in the blood-level of oxygen without risk of the complications which have been reported to develop occasionally at higher pressures. Oxygen poisoning, which in small animals leads to acute pneumonitis, does not appear to be a danger to man at this pressure, at any rate for short periods, and in 2 cases treatment for prolonged periods (48 hours continuously and 90 hours spread over a few days) has had no harmful effect. There is, of course, no risk to the patients (breathing oxygen) of caisson disease, which is due to nitrogen bubbles, while the risk to attendants (breathing air) is minimal if adequate time is allowed for decompression, and can indeed be abolished if oxygen is inhaled during the decompression process. The usual regulations pertaining to working in compressed air are complied with. The staff have chest films and periodic survey of ears, sinuses, and teeth roots.

PHYSIOLOGICAL CONSIDERATIONS

Oxygen is carried in blood in combination with hæmoglobin and also in simple physical solution in the plasma. Under normal conditions the hæmoglobin carriage is the more important. Normally in blood leaving the left heart the hæmoglobin is 95 per cent saturated, and with a hæmoglobin level of 15·3 g. per cent this represents an oxygen content of 19·5 vols. per cent. By contrast, the dissolved oxygen is approximately 0·25 vols. per cent, but though small in amount it is important in that it represents the immediate source available to the tissues.

The amount of oxygen in solution in the blood conforms to Henry's Law and depends on the solubility of the gas in plasma and upon its partial pressure in the gaseous mixture with which the blood is in equilibrium (i.e., the pulmonary alveolar content). In pure oxygen at 1 atmosphere pressure 100 mil. of blood at body temperature will take up 2·2 mil. of oxygen (Bohr, 1905). When the ambient pressure is doubled the amount of oxygen in solution is raised to about 4·2 mil. As Haldane showed (1895) in his

classical experiments on carbon monoxide poisoning this amount is about enough to supply the oxygen requirements of a resting mammal without hæmoglobin.

FIG. 251.—View into chamber from compressor and air-conditioning room.

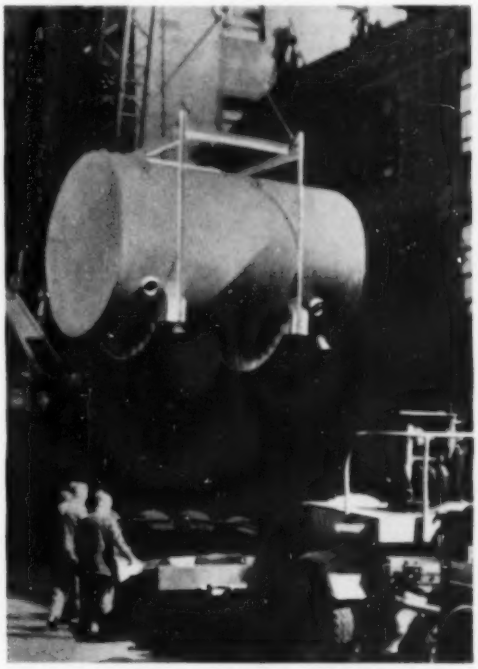

FIG. 252.—Hoisting chamber up on to roof site.

Raising the oxygen tension (and therefore the amount of oxygen in solution) confers another advantage—namely, that it increases the rate of transfer of the gas through the capillary wall to the tissue fluids and cells.

When air is breathed at normal atmospheric pressure the partial pressure of oxygen in mixed arterial blood is about 87 mm. Hg. When oxygen is breathed at twice the normal atmospheric pressure the partial pressure rises more than tenfold. Under such conditions, if a part of the body is deprived of its main blood-supply there develops a steep gradient between the increased partial pressure in the plasma and the greatly reduced pressure in the anoxic tissue (*Fig.* 253), so the rate of diffusion is correspondingly increased.

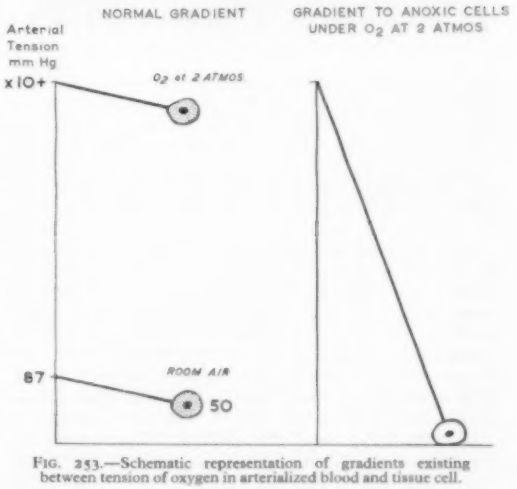

FIG. 253.—Schematic representation of gradients existing between tension of oxygen in arterialized blood and tissue cell.

CLINICAL AND EXPERIMENTAL OBSERVATIONS

During the past year the pressure chamber has been used for clinical observations on a variety of conditions in which the blood-supply to a part has been jeopardized, including arterial injuries, chronic obliterative arterial disease, and ischæmic amputation flaps. Detailed reports of illustrative cases will be given below.

Experimental observations have also been made on dogs, as a preliminary to clinical application, in three different fields. The original experiments on acute coronary obstruction have been extended, a series of observations has been made on the effect of temporary occlusion of the cerebral circulation, and, lastly, at the present time observations are in progress on the use of the chamber, combined with moderate hypothermia, for operations involving total cardiac arrest.

Finally, in a quite different field, the chamber has proved to be quite strikingly successful in the treatment of carbon-monoxide poisoning and in the cerebral anoxia of severe barbiturate poisoning.

Injury to the Main Artery of a Limb.—In 2 such cases treatment in the chamber is believed to have prevented massive gangrene of the affected part of the limb. The first was a young man aged 16 years, who sustained a Pott's fracture with compound dislocation at the ankle. The anterior and posterior tibial vessels and the peroneal vessels were divided, and the foot remained attached to the leg only by the tendons and their sheaths and two strips of skin little more than an inch across (*Fig.* 254). A mid-leg amputation seemed inevitable. However, one of us (G. S.) decided to try to preserve the foot. The cut ends of the torn posterior tibial artery were joined by suture, the dislocation was reduced, and the skin stitched. The cut ends of the posterior tibial vein could not be identified and venous blood leaked from the wound for the first two days. The patient was treated in the pressure chamber continuously for 48 hours and intermittently for the succeeding three days.

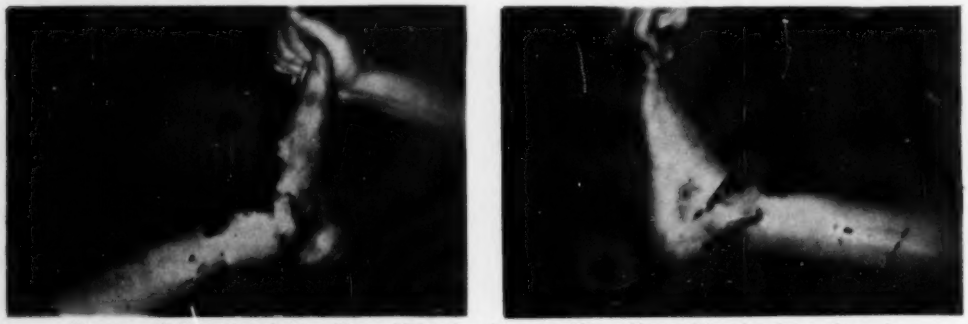

FIG. 254.—Medial and lateral views of foot and leg to show the extent of intact skin remaining after closure of wounds.

FIG. 255.—The condition of the foot up to 43 hours after repair.

Within a few hours the skin temperature over the foot rose and the whole foot assumed a normal colour (*Fig.* 255). At the present time (2½ months later) the toes are gangrenous, but the greater part of the foot is viable and it is thought that a transmetatarsal amputation may be effective.

The second case was a young woman aged 20 years admitted with fracture of the pelvis, rupture of one kidney, and comminuted compound fractures of both femora and tibia. The left leg was ischæmic, probably due to damage to the left femoral artery and pressure by a massive hæmatoma in the thigh. She was treated in the chamber for 15 hours. The left leg rapidly regained normal colour and temperature.

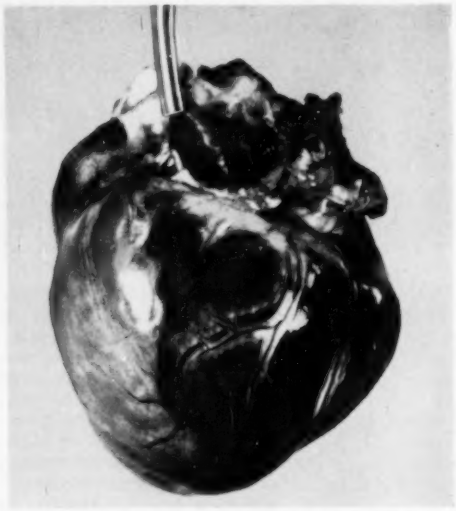

FIG. 256.—Dog's heart showing point of ligation of the left circumflex coronary artery. The muscle supplied by this branch has been injected with indian ink.

Now, 2 months later, only the toes are gangrenous. In both these cases although absolute proof is lacking our clinical experience suggests that massive gangrene was only averted by the use of the pressure chamber.

Peripheral Vascular Disease.—We have used the pressure chamber in 5 elderly patients with chronic occlusive vascular disease complicated by ischæmic neuropathy with unremitting pain, in 3 patients with ischæmic ulcers of the toes, and in 4 patients with early gangrene of the forefoot. These patients go into the chamber in groups of four or five, and stay there for 2–3 hours daily for up to 15 days. They were selected by reason of the severity and chronicity of their symptoms, and care was taken to make no

other change in their regimen or treatment that might influence their progress. All of the patients with ischæmic pain gained relief while they were in the chamber. At first pain recurred with increased severity on leaving the chamber, but later the relief was more prolonged, so that they were able to sleep without

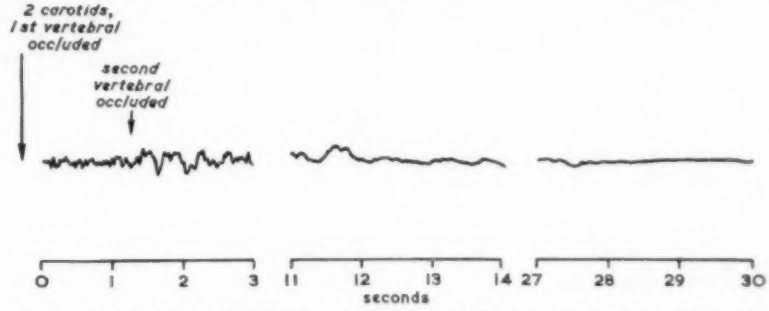

FIG. 257.—Electro-encephalographic tracings showing changes following occlusion of blood-vessels in the neck.

sedation. Since the pain had been present for a long period and had resisted previous care, we believe that the relief was in fact due to the treatment given.

In the other patients in this category the results were less impressive, but undoubtedly the colour of the viable skin proximal to the lesion improves while the patient is in the chamber, and we have gained the impression that demarcation of a gangrenous part occurs more quickly and that a distal amputation may sometimes be made feasible.

Coronary Artery Obstruction.—In their early experiments two of us (G. S., D. D. L.) showed the value of oxygen administration in a pressure chamber in the treatment of dogs with acute interruption of coronary blood-flow. These experiments have been extended to include a total of 50 dogs. In each case a ligature was placed around the circumflex branch of the left coronary artery (*Fig.* 256) so as to render about 40 per cent of the ventricular muscle mass ischæmic. As controls, 30 dogs were used in groups of 10. Dogs in group 1 breathed air at normal atmospheric pressure, dogs in group 2 breathed oxygen at normal pressure, while dogs in group 3 breathed air at 2 atmospheres. In each of these groups about half the dogs developed ventricular fibrillation and died within the experimental period of 2 hours, the mortality over the 3 groups being 56 per cent.

Table I.—SHOWING RESULTS OF LIGATION OF LEFT CIRCUMFLEX CORONARY ARTERY UNDER DIFFERENT ENVIRONMENTAL CONDITIONS

No. of Dogs	*Atmosphere Breathed*	*Deaths*
10	Room air	6
10	O_2 at 1 atmos.	6
10	Compressed air at 2 atmos.	5
20	O_2 at 2 atmos.	2

The remaining 20 dogs were placed in the pressure chamber immediately after the ligature had been applied and oxygen was administered at 2 atmospheres pressure. In this series only 2 dogs (10 per cent) died within the experimental period (*Table I*).

While the artificial conditions of this experiment cannot be compared directly with the clinical state of acute coronary occlusion, the experimental observations seem to justify a clinical application and this we propose to undertake in the near future. Most of the dogs in the control series died with ventricular

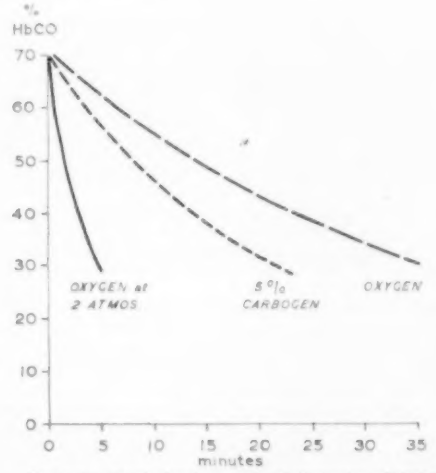

FIG. 258.—Graph showing speeds of elimination of HbCO from mixed venous blood with the animals breathing oxygen at 1 atmosphere, 5 per cent carbogen at 1 atmosphere, and oxygen at 2 atmospheres pressure.

fibrillation, and this is a common feature in deaths from coronary occlusion in man. It seems possible therefore that the protection offered by oxygen administration in the chamber might tide over this phase to enable a collateral circulation to develop. Moreover the improved oxygen supply might ensure survival of a greater part of the myocardial cell mass, and lessen the risk of later hypodynamic heart failure.

Cerebral Vascular Occlusion.—Experiments have been carried out (G. S., D. D. L., I. McA. L., and G. R. S.) to determine whether the administration of oxygen in the pressure chamber may be of value during operations involving temporary arrest of the cerebral circulation. In two series, each of 5 dogs, both common carotid arteries and both vertebral arteries were occluded by snares low in the neck.

Electro-encephalographic tracings were obtained from cortical electrodes. The dogs in group 1 breathed air at normal atmospheric pressure, those in group 2 breathed oxygen in the chamber at 2 atmospheres pressure. In the dog the collateral circulation, mainly through the heavy muscles of the nape of the neck, is so great that even this fourfold ligation does not cause immediate death. However, in the control dogs the electro-encephalographic of arrest, permitting survival with no neurological or other sequelæ, has been 35 min.

In applying this type of experiment to man we encounter the difficulty that there is no warning signal when the safe period is nearing its end. We shall have to proceed with great caution, therefore, to determine what can be done without risk. It is too soon yet even to hazard a guess as to the scope of the method in open heart surgery.

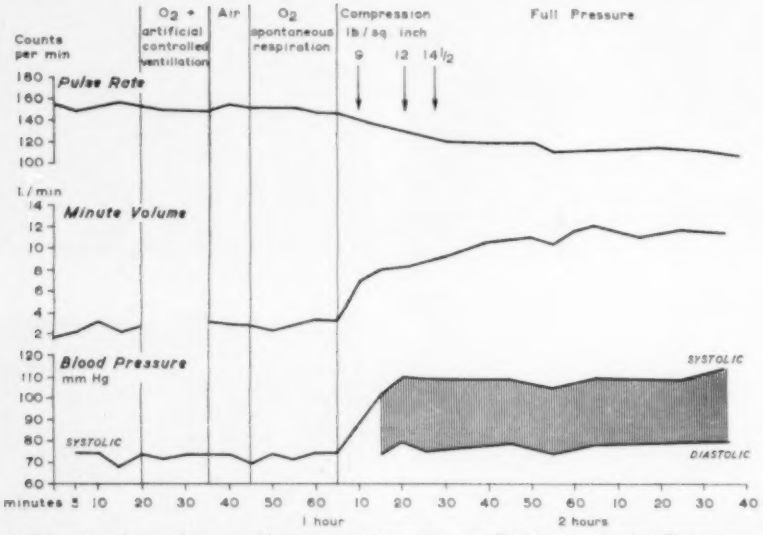

FIG. 259.—Pulse-rate, minute volume, and blood-pressure changes in a case of barbiturate poisoning. Oxygen was administered at room pressure for 1 hour. The pressure was then raised to 2 atmospheres, with immediate benefit.

record showed cessation of cortical activity starting within 15 sec. of occlusion of the main vessels (*Fig.* 257). At this point the snares were loosened to restore the circulation and the procedure repeated with the animal breathing oxygen at 2 atmospheres. This time there was no loss of cortical activity during the ensuing 30 min. In the 5 dogs of group 2 treated in the chamber from the beginning, the electro-encephalograph remained normal throughout.

These experiments, which will be reported in detail elsewhere (Smith, Lawson, Renfrew, Ledingham, and Sharp, 1961), give support to the view that the pressure chamber may be of value during operations on the cerebral circulation in man.

Total Circulatory Arrest.—Experiments are at present being carried out to test the hypothesis that tissues overloaded with oxygen may withstand total circulatory arrest for longer periods than normal, and to determine the maximum safe period of arrest when combined with moderate hypothermia. The method being used in dogs is to induce hypothermia by surface cooling until the mid-œsophageal temperature reaches about 28° C. and then to produce total arrest by clamping the superior and inferior venæ cavæ, the aorta, and the pulmonary artery. At the end of the agreed period the clamps are released, the wound is closed, and the animal's progress watched. In successive experiments the period of arrest is lengthened. So far, the maximum safe period

Carbon Monoxide Poisoning.—The value of oxygen administration at raised ambient pressure in the treatment of carbon monoxide poisoning was demonstrated by Haldane. Experimental work from this department (Lawson, McAllister, Smith, 1959, 1961) confirmed the efficacy of the treatment, and more recently its clinical application has been reported (Smith and Sharp, 1960; Smith, Sharp, and Ledingham, 1961).

Up to the present time 18 cases of carbon monoxide poisoning have been treated with complete success, including several who on clinical grounds appeared to be moribund on admission.

In this condition treatment in the chamber serves a double purpose, for the increased oxygen content of the blood serves to maintain life during the critical phase, while the increased partial pressure helps to drive off poisonous gas. Under treatment in the chamber the carbon monoxide content of the blood falls almost to zero within ½ to 1 hour and consciousness is quickly regained. Striking features are the absence of myocardial and cerebral sequelæ and the general well-being of the patients immediately after the treatment is concluded. This method is superior to all the more usually available techniques of resuscitation (*Fig.* 258).

Barbiturate Coma.—While most patients with barbiturate poisoning respond well to conventional measures, there arises occasionally a state of

medullary depression which may be due to cerebral anoxia.

One such patient, a woman aged 22 years, was admitted deeply comatose after taking about 20 nembutal capsules. There was mild spasticity of the upper limbs, the pupils did not react to light, and the deep reflexes were absent. Gastric lavage was performed and megimide administered. The blood-pressure was 110/75 mm., the pulse regular and of fair quality, its rate 95 per min. Twelve hours later she was still comatose and did not react to painful stimuli. The pupils still failed to react. Respiration was shallow and infrequent, 3 or 4 per min. The colour was ashen grey, with cyanosis of lips and ear lobes. The pulse was thready and almost imperceptible at the wrist. The systolic pressure was about 75 mm. Hg. Oxygen was given on a respirator at normal atmospheric pressure, without benefit. She was then taken to the chamber, still breathing oxygen, and the pressure raised to 2 atmospheres. The changes in pulse-rate, minute volume of inspired gas, and blood-pressure are shown in *Fig.* 259. The improvement was maintained after removal from the chamber and the later course was uncomplicated.

CONCLUSIONS

Up to the present time we have made use of the pressure chamber in an exploratory way for a wide variety of conditions, some of which we have described in detail above. A good deal of further experience will be required to assess its value in all these different circumstances.

This much we have proved, that using the chamber causes far less inconvenience and discomfort than had been expected and none of the dangers which have sometimes been feared.

A few members of the staff have found it difficult to adjust pressures within the middle ear and sinuses. Certainly anyone with sinusitis should avoid using the chamber. Up to the present time none of our patients has encountered such difficulties. In anæsthetized and comatose patients there has been no case of rupture of the ear drum. On one occasion a member of the staff, after having been in the chamber several hours, was decompressed rapidly, and subsequently developed multiple joint pains which were attributed to trapped nitrogen. This has been the only instance of the decompression syndrome, and it can readily be prevented by oxygen administration during the decompression process.

No case of oxygen poisoning has occurred in our experience. It has long been known that oxygen at high ambient pressure is toxic to small animals, but in man at 2 atmospheres pressure the risk appears slight. We have had patients under treatment for 48 hours continuously and for over 90 hours at intervals in the course of a few days with no evidence of toxic damage. The attendants, who breathe air, are, of course, not subject to this hazard.

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SHORT NOTES OF RARE OR OBSCURE CASES

RETROGRADE JEJUNOGASTRIC INTUSSUSCEPTION FOLLOWING PARTIAL GASTRECTOMY

By J. K. M. RAWLINSON

FORMERLY SENIOR SURGICAL REGISTRAR, LIVERPOOL ROYAL INFIRMARY

My object in reporting this case is twofold: firstly, to describe an unusual complication occurring eight years after partial gastrectomy; and, secondly, because the march of events leading up to the emergency operation forms an interesting clinical study which should perhaps have led to a correct pre-operative diagnosis.

CASE REPORT

This patient, a man aged 58 years, had had a partial gastrectomy at another hospital for a duodenal ulcer, after a dyspeptic history of thirty years. This gastrectomy was subsequently found to be antecolic in type, with afferent loop to lesser curve and full-width gastric stoma, i.e., no valve had been constructed. From his description he had suffered from a typical afferent-loop syndrome ever since this operation, consisting of the vomiting, shortly after a meal, of bile with no food particles. This had occurred regularly once every 2–3 days up to the time of his present illness. Various investigations had been carried out, and conservative treatment tried without much success.

He was admitted as an emergency with abdominal pain and vomiting. This had started suddenly some

twenty-four hours earlier, with severe, constant, epigastric pain passing through to the back, but gradually becoming easier. It was associated with profuse vomiting. The vomit was yellow-brown in colour, not fæcal but clearly of upper intestinal content. His bowels had been opened the previous evening, but not on the day of admission, though

FIG. 260.—Diagram illustrating operative findings.

he was passing flatus. His weight was steady, but he had never regained his pre-gastrectomy weight.

ON EXAMINATION.—His temperature was normal, pulse 70/min., and blood-pressure 150/70 mm. He was pale and dehydrated. The tongue was clean but dry. There was marked tenderness in the epigastrium and left hypochondrium, with guarding, and a suggestion of some thickening just above and to the left of the umbilicus. There was no distension, no visible peristalsis, and no free fluid; bowel-sounds were present, though diminished. Hernial orifices were intact and rectal examination was negative.

No definite diagnosis was made. The patient was thought to be suffering from upper small-bowel obstruction of unknown aetiology, or possibly acute pancreatitis, or an acute exacerbation of a stomal ulcer. A straight radiograph of the abdomen was of no help, showing no gas shadows, no fluid levels, and no distended bowel. The serum-amylase was normal. As he seemed to be improving it was decided to keep him under observation and in the meantime correct his fluid and electrolyte balance.

The position was reviewed some 12 hours later. There had been no change in temperature, pulse, or blood-pressure. The pain, if anything, was easier, though it was still present and still constant, rather than colicky. Large amounts of fluid had been aspirated from the stomach. At first this had been yellow-brown, as before, but it had now become faintly blood-stained. Abdominal examination showed little change in the physical signs, except that he was not quite so tender in the left hypochondrium, and what had before been a rather indistinct thickening was now a more definite mass just above and to the left of the umbilicus. A further straight radiograph of the abdomen showed nothing new.

It was clear that there was still obstruction, and arrangements were therefore made for laparotomy. The blood-staining of the gastric aspirations became more marked, and by the time of operation almost pure blood was being withdrawn from the gastric tube.

AT OPERATION.—Under the anæsthetic it was possible to define the mass more clearly. This was approximately 6 in. × 3 in., with the long axis running upwards and to the left from the umbilicus.

The abdomen was opened through the previous mid-line incision. There were many adhesions. When these had been divided, the gastrojejunostomy was displayed, antecolic, with the afferent loop passing up to the lesser curve. It was seen that the efferent loop had intussuscepted in a retrograde manner; the apex of the intussusception was approximately 12 in. from the stoma, and had passed proximally up the efferent loop, eventually passing through the stoma into the stomach. A total of some 6 in. of jejunum (3 in. in both directions) lay within the stomach (*Fig.* 260). The intussusception was reduced with some difficulty by pressure from above through the wall of the stomach, and gentle traction from below. After reduction a split approximately 2 in. long was found in the long axis of the bowel about 12 in. below the stoma, i.e., close to the probable site of the apex of the intussusception. This presumably had occurred during the manipulations necessary to effect reduction.

No cause could be found for the intussusception. Apart from considerable œdema and congestion, nothing abnormal could be seen or felt in the involved bowel. The split in the wall of the jejunum was repaired transversely in two layers, and the abdomen closed.

It is interesting to note that the gastrojejunal anastomosis appeared to be quite normal, and the afferent loop did not appear unduly dilated or hypertrophied. There was no obvious kinking, although many adhesions were divided before the area of the anastomosis could be displayed, and it is possible that these may have been partially obstructing the afferent loop close to the stoma.

Recovery was uneventful except for a small area of collapse in the left lower lobe. When seen two months later all was well. The patient was eating without discomfort, and had had very much less bilious vomiting; whereas previously he would vomit regularly every 2–3 days, he had only vomited three or four times in the two months since his discharge.

DISCUSSION

It is interesting to look back over the clinical picture in the light of the subsequent findings. The march of events is quite clear, although at the time the significance of the symptoms and signs was not correctly appreciated.

The sudden onset of severe upper abdominal pain accompanied by profuse vomiting of upper small-bowel contents indicated a high obstruction. The tenderness in the upper abdomen with its associated guarding made the palpation of a mass uncertain, but it is likely that the thickening palpable at the first examination was in fact the intussusception. (According to Irons and Lipkin (1955) jejunogastric intussusception starts as a jejunojejunal intussusception, the apex eventually passing through the stoma into the stomach. The thickening palpable at this stage was probably the intussusception while it was still jejunojejunal.) Some 12 hours later pain and tenderness, though still present, were less marked, so that voluntary guarding was less. A more definite mass was now palpable with its long axis extending upwards and to the left from the umbilicus; no doubt the intussusception was larger having now passed through the stoma into the stomach. The gastric aspirations were still profuse because there was still obstruction, but now they contained blood in increasing quantities, characteristic of the later stages of intussusception. Thus the three characteristic features of jejunogastric intussusception had developed gradually over a period of 36 hours—namely, high intestinal obstruction, a palpable mass above and to the left of the umbilicus, and hæmatemesis—and they had developed in the order in which they might have been expected.

The cause of jejunogastric intussusception is unknown. Just over a hundred cases have been reported (Madding, McLaughlin, and de Riemer, 1956), and in none has any lesion of the jejunum been demonstrated as a possible precipitating factor. Reverse peristalsis does occur normally in the jejunum, and

it is thought that the intussusception starts as an exaggerated or deranged form of this. Marked reverse peristalsis has been observed in the upper jejunum after acid gastric contents have passed into it (Aleman, 1948). It has also been suggested that the dilatation and hypertrophy of the jejunum immediately distal to the stoma which commonly occurs after gastrojejunal anastomosis may predispose to abnormal or disordered peristalsis. Another possible precipitating factor is the presence of adhesions in the region of the stoma, producing kinking, or interfering with the normal progress of peristaltic waves. In this patient the jejunum distal to the anastomosis was not hypertrophied or dilated, but there were many adhesions in the area.

It is interesting to note that this patient described a typical afferent loop syndrome which had been present ever since his gastrectomy and which, in the first two months after the present operation, was greatly improved. It is attractive to suggest that whatever was causing this also precipitated the intussusception, and it may be that the adhesions were responsible for both conditions.

Jejunogastric intussusception occurs over a wide age-group, but it is commoner in women than in men. It is seen after both gastro-enterostomy and partial gastrectomy, although more frequently after the former (13 : 1). It does not appear to bear any relation to the size, position, or direction of the stoma (Foster, 1956). It may occur at any time from the immediate post-operative period up to thirty years later (Coates, 1949). The case reported here occurred eight years after partial gastrectomy. Aleman (1948) has described three types, viz.:—

1. Descending (10 per cent), afferent loop passing through the stoma;
2. Ascending (74 per cent), efferent loop passing backwards through the stoma; and
3. Combined (16 per cent), both afferent and efferent loops passing together through the stoma.

Types 1 and 2 are usually late complications, while Type 3 occurs most commonly in the early post-operative period.

The condition occurs most often in the acute form as illustrated here, the salient clinical features being those of a high intestinal obstruction, with hæmatemesis and a palpable mass. A chronic form is recognized, however, consisting of recurring attacks of vomiting of upper small-intestinal contents, usually soon after a meal, and accompanied by cramp-like epigastric pain. In these cases a barium meal may demonstrate the intussusception passing into the stomach.

A corrective pre-operative diagnosis is rarely made. A palpable mass should give a valuable lead, though in 50 per cent of cases (Foster, 1956) nothing can be felt in the abdomen. Vomiting is always present, usually with some blood. It is as well to bear the condition in mind in cases of hæmatemesis following gastro-enterostomy. Such cases are usually treated conservatively at first, but if, in fact, the cause is an intussusception, valuable time may be lost before advising operation, and the chances of a successful outcome seriously endangered. Conditions which should be considered in the differential diagnosis are acute pancreatitis, recurrent or anastomotic ulceration, or high small-bowel obstruction (all of which were thought to be possibilities in the case here described). The condition is so rare, however, that it is not usually considered until it is actually seen at operation (Moroney, 1947).

Treatment is always surgical and should be undertaken without delay. Mortality in cases where operation is delayed 48 hours or more after the onset of symptoms is over 50 per cent, but it is only 10 per cent if undertaken sooner (Aleman, 1948). Conservative treatment carries a mortality of 100 per cent (Lawson and Whitener, 1950). The intussusception is rarely gangrenous and can usually be reduced easily. Recurrence of intussusception is almost unknown, and no special precautions are necessary to prevent this. As the intussusception may occur after gastrectomy as well as after gastro-enterostomy, it would seem pointless to convert a gastro-enterostomy into a gastrectomy in order to prevent a recurrence, always providing, of course, that the gastro-enterostomy is working well.

As has been stated, the condition is only rarely seen; at the same time, if it is missed for too long, a fatal outcome is more than likely. If a patient who has had a partial gastrectomy or a gastro-enterostomy develops sudden severe epigastric pain, accompanied by vomiting which may be blood-stained, jejunogastric intussusception should be considered as a possible cause. If a mass is palpable, the diagnosis is probably correct and surgical treatment should be instituted forthwith.

SUMMARY

1. A case of retrograde jejunogastric intussusception following partial gastrectomy is described.
2. The causes and characteristics of the condition are discussed and the inherent dangers emphasized.
3. The literature is briefly reviewed.

I would like to express my thanks to Professor C. A. Wells and Mr. I. MacPhee for permission to publish this case, and to Miss Barbara Duckworth for the diagram.

Addendum.—Within a month of this occurrence another patient under investigation in the same ward precipitated a similar retrograde intussusception by straining at stool. He had had a gastro-enterostomy for many years and was admitted on account of attacks of pain which recurred at intervals of several months. The pain he had immediately prior to his operation for intussusception was of a similar character, though more severe.

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PRESACRAL LIPOMATA

By A. P. MOWAT and C. G. CLARK
FROM THE ABERDEEN ROYAL INFIRMARY

Lipomatous tumours lying behind the rectum are rare. Pre-operative diagnosis is difficult and surgical removal, often hazardous, may be unrewarding, for fatty tumours arising from the posterior abdominal wall and pelvis are often sarcomatous. We record a case of a benign tumour in this region, noting the rarity of the condition and the difficulties encountered in its management.

CASE REPORT

H. S., a housewife, aged 61 years, was admitted to hospital in June, 1958, for investigation of post-menopausal bleeding, later attributed to senile vaginitis, with cervical erosion. A soft fixed mass was palpated in the pelvis and laparotomy was performed on June 11, 1958. A tumour was found behind the rectum but was not removed because of its inaccessibility and apparent benign nature.

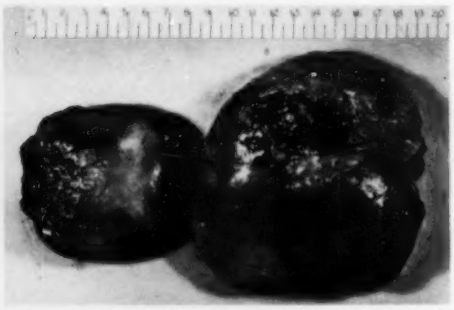

Fig. 261.—Photograph of the tumours removed from the presacral region in the present case.

In May, 1959, she was admitted to Aberdeen Royal Infirmary as her family doctor considered that the mass had increased in size. It was difficult to assess whether she had any symptoms referable to the tumour. She complained of some looseness of stool with mucous discharge, but this symptom had been noted intermittently for twenty years. When present it was accompanied by vague left-sided abdominal pain suggesting a long-standing mucus colitis.

On Examination.—She was a healthy woman and physical signs were confined to the abdomen. There was a right lower paramedian scar. The rectum was pushed forward by a large, smooth, firm mass lying in the hollow of the sacrum. A needle biopsy consisted of necrotic debris containing clumps of cells resembling epithelial cells, but no diagnosis could be made on the sample obtained. A barium enema showed only narrowing of the rectum due to an extrinsic cause and a solitary diverticulum of the sigmoid.

A second laparotomy was performed on June 4, 1959. A large mass filled the pelvis, extending to the pelvic brim and displacing the rectum forward. The peritoneum on the left side of the rectosigmoid junction was opened and, after dividing the inferior hæmorrhoidal vessels, the upper part of the mass was defined. A well-encapsulated tumour was readily enucleated by blunt dissection before it was appreciated that a second tumour lay below it. Both tumours were adherent to the sacrum, the lower one particularly so, and after enucleation a bare area of bone was exposed. Following removal of the second tumour there was considerable bleeding which could only be controlled by packing. Blood was transfused rapidly to restore the resulting acute hypotension. On removing the pack a little bleeding persisted, and since accurate definition of the vessels was impossible, oxycel gauze was placed in the tumour bed. The wound was closed without drainage.

On the fourth post-operative day the patient again became hypotensive but recovered with transfusion. It seems likely that she suffered a second hæmorrhage into the tumour bed, for subsequently there was mild icterus (serum-bilirubin 1·7 mg./100 ml.) and her Hb percentage fell. A pelvic abscess then developed and required drainage through the posterior rectal wall on June 13. She was finally allowed home on her thirty-fifth post-operative day.

The Specimens (*Fig.* 261) consisted of two rounded masses, one measuring 10 × 10 × 7 cm., weighing 370 g., and the other measuring 8 × 8 × 5 cm., weighing 120 g. On transection both showed the same general appearance of adipose tissue with widespread hæmorrhage and congestion. Histologically both showed the features of partially strangulated benign lipomata and were presumably lobules of the same tumour. The explanation of the strangulation was not apparent microscopically.

The patient was seen at intervals during the next few months and an incisional hernia developed. Because of reports in the literature suggesting that lipomatous tumours of this nature can recur and assume malignant characteristics, it seemed advisable to repair the hernia and re-examine the pelvis during operation.

On Jan. 26, 1960, the incisional hernia was repaired. There was no sign of tumour recurrence in the pelvis, which was remarkably free of any indication of previous operation or sepsis. The patient was in excellent health and with a normal hæmoglobin. Curiously she has had no recurrence of her colonic symptoms since the removal of the lipomata. She was last seen on July 13, 1961, and was in good health, free from any recurrence of the original condition.

COMMENT

A benign presacral lipoma is reported by Castleman (1958). Boyd (1925) and Adair, Pack, and Farrior (1932) describe liposarcoma in this situation. DeWeerd and Dockerty (1952) suggested that tumours in the posterior wall of the pelvis should be included in the classification of retroperitoneal neoplasms. They emphasize the fact that, although lipomas are generally benign, when situated in the retroperitoneum they are frequently malignant (60 per cent). Pemberton and Whitlock (1934) found that only 20 per cent of retroperitoneal fatty tumours were malignant, but Farbman (1950), reviewing the literature, noted that of 14 tumours originally reported as benign, 6 recurred and 3 of the recurrences showed sarcomatous change.

The proximity to vital structures makes even the benign lipomas difficult to remove, and hæmorrhagic shock and infection are common (DeWeerd and Dockerty, 1952). The operative mortality in this condition appears to be between 8 per cent and 15 per cent.

The present case illustrates the diagnostic difficulties encountered with a presacral lipoma, and, in considering the available evidence, certain features in the management of these cases emerge. In this patient the tumour was discovered initially during examination for an unrelated complaint. Of the lesions to be considered in the differential diagnosis of a fixed swelling in this region, tumours of the pelvic organs, sacrococcygeal chordoma, chondrosarcoma, and tuberculosis of the sacrum may be mentioned. Gentil and Coley (1948) suggest that needle biopsy is a useful diagnostic tool in these circumstances, though it was of no assistance in the present case probably owing to the inadequacy of the biopsy.

We must emphasize the danger of regarding a smooth rather inaccessible pelvic tumour as benign, especially when no biopsy is performed. This error of judgement occurred at the patient's initial laparotomy and is understandable when we consider the rarity of the condition and the paucity of clinical reports. There is no doubt that the tumour under discussion was increasing in size (a feature confirmed on examination by the first surgeon). If classified with retroperitoneal lipomata the risk of malignant change is high and early removal is indicated.

The smooth encapsulation of the present tumour facilitated its complete removal, though the subsequent blood-loss was difficult to control, endangering the patient's life. Such experience is not uncommon in removing this type of lesion, more particularly the lobulated variety which tends to infiltrate between tissue planes. It is a matter of conjecture whether the features described as due to partial strangulation in the specimen resulted from manipulation during operation or from hæmorrhages into the tumours. It is clear, however, that the tumours were more vascular than their counterparts elsewhere in the body. Due consideration should be given to the advisability of internal iliac artery ligation in similar circumstances.

Although the occurrence of an incisional hernia provided an opportunity to re-explore the abdomen in this patient, the evidence of Farbman (1950) suggests that in similar circumstances there is justification for a 'second look' operation (Wangensteen, Lewis, Arheiger, Muller, and MacLean, 1954). The relatively frequent benign recurrences can be dealt with, and in a number of recurrent lesions where sarcomatous change has resulted early excision is facilitated.

SUMMARY

A case of benign presacral lipoma is recorded because of its rarity. Experiences encountered in dealing with this problem are discussed, with reference to the vagaries of retroperitoneal lipomata with which it may be classified. The conclusion that relatively inaccessible and apparently benign fatty tumours in the pelvis should be removed is warranted.

The natural history of these tumours lends support to the suggestion of performing an exploratory operation some months after removal of the primary tumour.

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CALCIFIED 'LATERAL ABERRANT THYROID'

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IT is now generally accepted that the 'lateral aberrant thyroid' represents a metastatic deposit in a cervical lymph-node, arising from a primary focus in the thyroid gland. The primary carcinoma is often discovered only after serial section of the gland.

There have been in recent times several papers supporting this viewpoint (Wozencraft, Foote, and Frazell, 1942; Frantz, Forsythe, Handford, and Rogers, 1942; Willis, 1953; and Dalgaard and Wettland, 1956). Reference has been made to 'calcified spherules' or psammomatous bodies (Willis, 1953), but there is no report of extensive calcification involving the 'lateral aberrant thyroid'.

This communication records one such case-history, and adds this condition to the differential diagnosis of calcified lateral cervical lesions.

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CASE REPORT

S. F., a 73-year-old housewife, who gave a poor history, was admitted on June 1, 1959, for treatment of a swelling in the left side of her neck which she had had for the past eight years. She thought it was gradually increasing in size, and it caused her some pain. There had been no attacks of inflammation and no discharge from the mass. She was hypertensive and suffered from fairly severe osteo-arthritis of both knees and both hips. Inquiry into her past history revealed that she had had a right femoral hernia repaired in May, 1955, and she had been treated conservatively for cystitis in November, 1957. Intravenous pyelography, at this stage, demonstrated a normal renal tract, but there was some calcification in the left hypochondrium, considered by the radiologist to be in the splenic artery.

ON EXAMINATION.—Short, obese, elderly female subject. Mouth edentulous; gums and tongue healthy. In the left *posterior* triangle of the neck there was a stony, hard mass approximately 3 cm. in diameter; its surface was finely lobulated and it was painless. It was situated

subcutaneously and was freely mobile, being unattached to skin or deep structures. There were a few small mobile lymph-nodes in both upper deep cervical chains.

The left lobe of the thyroid gland was very slightly enlarged, especially the lower pole. In addition the patient had a small sebaceous cyst of the scalp overlying the left occipital bone.

FIG. 262.—Photograph of neck showing position of incision, placed over the swelling. Note that the sternomastoid is anterior to the scar.

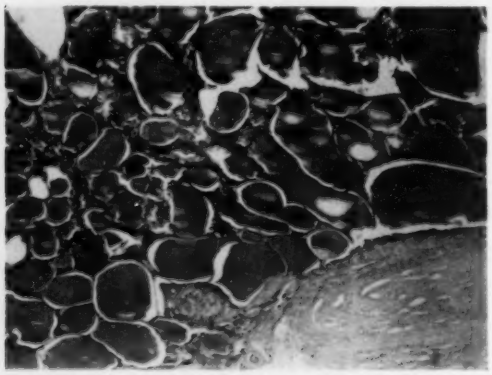

FIG. 264.—Photomicrograph of section of specimen showing well-defined thyroid follicles and thick fibrous capsule. (× 88·5.)

General examination revealed fairly prominent hypertension (blood-pressure 235/160) with cardiomegaly. Lung fields were clear. Abdomen—gross obesity; no abnormality detected.

Radiography of the chest showed cardiomegaly and a calcified lesion on the left side of the neck.

Pre-operative diagnosis: calcified tuberculous glands. In view of the great mobility of the swelling, a calcified lipoma was considered as an alternative.

AT OPERATION (June 2).—Local anæsthesia was employed. The sebaceous cyst was easily removed. The mass in the neck was displayed through a skin-crease incision placed over its maximal convexity (*Fig.* 262). It was found to be an incompletely calcified mass which 'shelled out' very readily (*Fig.* 263). The wounds healed by first intention and the patient was transferred to a convalescent home for social reasons.

PATHOLOGICAL REPORT.—"Nodule 35 × 20 × 25 mm., which is homogenously calcified. The greater part of the nodule is thyroid tissue consisting mainly of follicles filled with colloid, and lined by flattened cells. No lymphoid tissue recognizable. Surrounding the thyroid tissue is a thick layer of calcified fibrous tissue. In view of the fact that this thyroid nodule lay in the posterior triangle of the neck, it must be regarded as a metastasis of a well-differentiated follicular carcinoma of thyroid." (*Fig.* 264.)

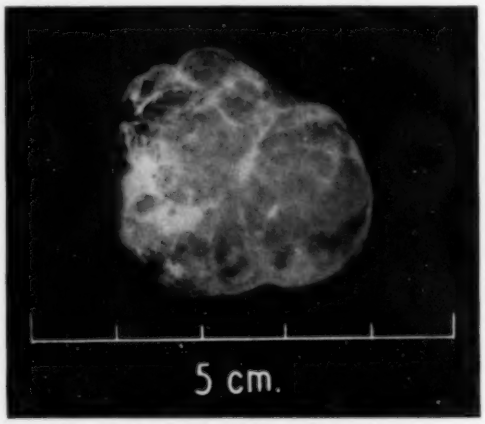

FIG. 263.—Radiograph of removed specimen.

After receipt of this report, I^{131} uptake and scanning studies were performed. These showed a normal uptake by the gland as a whole with a slightly diminished uptake by the left lobe as compared with the right. It was proposed to explore the thyroid gland, but the patient refused further operation. A body scan was arranged but the patient failed to attend on the appointed day and it was considered that no further radio-isotope should be administered for a repeat scan.

The patient was subsequently admitted to an old peoples' home on the coast, and the last report obtained stated that she was alive and well on March 15, 1960.

SUMMARY

An unusual case (? the first) of gross calcification in an 'aberrant' thyroid gland is described.

The theory that this thyroid tissue arises from the ultimobranchial body is not nowadays accepted. Rather it is a metastatic deposit from, what is often the case, a microscopic carcinoma of the thyroid gland.

I wish to express my thanks to Professor Ian Aird for permission to treat this patient and to report the case-history.

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COLIC INTUSSUSCEPTION DUE TO A CHRONIC GRANULOMA

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CASE REPORT

Mrs. O. W., aged 41 years, was referred in November, 1958, with a history of diarrhœa and intermittent colicky abdominal pain for the preceding ten weeks. She was passing 4–6 fluid motions daily, with occasional mucus and traces of blood.

Clinical examination of this very obese patient at first revealed no abnormality. A week later, however, following a sigmoidoscopy, the abdomen was palpated again, and a mobile painless tumour was felt in the mid-epigastrium.

Fig. 265.—Barium enema demonstrating chronic intussusception.

A barium enema (Dec. 2) was reported on as showing the appearance of an obstruction of the mid-transverse colon consistent with a chronic intussusception or a neoplasm (*Fig.* 265). After suitable pre-operative preparation of the bowel with phthalylsulphathiazole and neomycin an exploratory laparotomy was performed on Dec. 9. An irreducible colocolic intussusception of the proximal half of the transverse colon was found. The proximal bowel was distended and distal bowel collapsed. A right hemicolectomy was performed, continuity being re-established with an end-to-side ileo-transverse anastomosis.

The patient's recovery was uneventful and she was discharged from hospital on Dec. 24.

Operation Specimen.—The opened bowel revealed the point of intussusception, an elongated sausage-shaped mass about 5 cm. in length. Its base was wide and the underlying bowel was pale, thickened, and œdematous. The apex of the mass was necrotic and ulcerated. There was no enlargement of the regional lymph-nodes in the mesocolon and the bowel otherwise appeared normal (*Fig.* 266).

Histology.—Microscopy revealed a diffuse lesion with occasional more demarcated foci. All layers of the bowel were thickened, with capillary congestion and proliferation of vascular endothelium. Scattered throughout the interglandular tissue were many plasma cells and a few eosinophils (*Fig.* 267). There were also areas of superficial ulceration. The subserosa showed marked swelling and fibroblastic reaction with inflammatory cells.

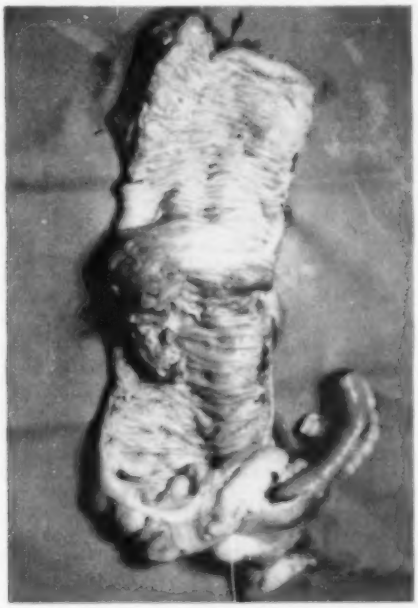

Fig. 266.—Operation specimen showing polypoid granuloma.

The general background appeared to be a marked fibroblastic proliferation in œdematous connective tissue. A few foreign-body giant cells and histiocytes were seen.

COMMENT

The main features of this tumour would seem to conform to the term 'non-specific granuloma'. Although eosinophils were present in the cellular infiltration, they were not in such predominance as to justify the term 'eosinophilic granuloma.' Apart from this, the histological pattern conforms to the pathological criteria indicated by Virshup and Mandelberg (1954) as necessary for making such a diagnosis, viz.:—

1. A granuloma composed of cells of histiocyte or fibroblastic derivation in which eosinophils predominate.
2. Marked œdema of tissue.
3. Fibroblastic proliferation as a constant background.

4. The occasional occurrence of giant cells of the foreign-body type.

5. The frequent occurrence of necrosis.

In a review of 205 cases of colonic intussusception from Swiss Clinics, by Babaiantz and Bugnion (1953),

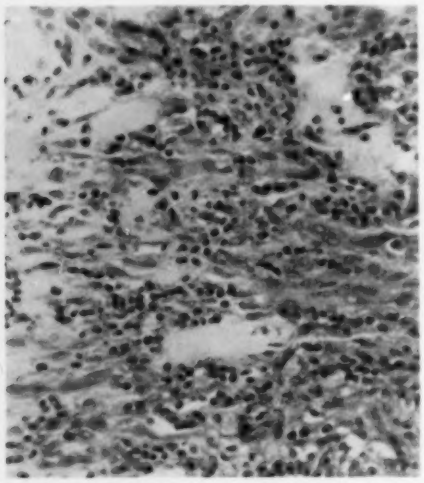

FIG. 267.—Photomicrograph of granuloma demonstrating widespread cellular infiltration. (× 200.)

only 9 cases were found to be of colocolic variety. The symptoms are liable to be less acute than in small-bowel intussusception. As indicated by Aird (1949), the lumen of the sheath is wide, the walls of the intussusception are thin and less vascular than ileum, and the mesentery more slender, so that vascular changes develop slowly. In this patient, symptoms had been present for nearly three months before operation.

The radiological findings in colocolonic intussusception are typical. In the acute case plain films of the abdomen may demonstrate, in addition to the appearance of intestinal obstruction, the following features which are characteristic of the condition:—

1. The soft-tissue shadow of the intussusception may be visible in the right side of the abdomen, with a relative absence of gas shadows.

2. The apex of the intussusception may be outlined as a convex filling defect in the gas shadow of the colon distal to it.

In the chronic case, such as the one now reported, the patient is more likely to be referred for routine barium enema as the first radiological investigation.

In a typical case the barium enema appearances may be as follows:—

1. *Obstruction to the Enema.*—The flow of barium is arrested by the apex of the intussusceptum (*Fig.* 265).

2. *Filling Defect.*—The apex of the intussusceptum projects into the head of the barium, causing a characteristic 'cupping' at the site of the obstruction (*Fig.* 265).

3. *Filling of the Intussusception.*—After the initial sudden arrest of the flow of barium, a small column of barium insinuates into the fold between the intussusceptum and intussuscipiens.

This gives rise to the characteristic 'lobster claw' appearance. Alternatively, this thin layer of barium between the sheaths of the intussusception may have a more widespread 'coiled spring' appearance. This finding is typical of the more chronic type of intussusception and is due to the barium coating the folds of bowel caused by the reefing effect of the pull of the mesentery.

We wish to thank Dr. D. Fulton, of Peterborough, and Dr. C. H. G. Price, of Bristol, for their advice on the histological pattern of this lesion.

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CONGENITAL DIAPHRAGM OF THE DUODENUM CAUSING INTESTINAL OBSTRUCTION IN AN ADULT

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THIS case demonstrates a rare cause for high intestinal obstruction in an adult. Congenital duodenal obstructions are classified as intrinsic or extrinsic. A perforated web is probably the only intrinsic variety capable of remaining asymptomatic until adult life.

CASE REPORT

D. S., a commercial buyer, aged 37 years. He was admitted as a semi-emergency on Dec. 26, 1959.

HISTORY.—Before the present episode the patient had regarded his digestion as perfectly normal. In June, 1959, he had an episode of pain and vomiting, which he described as 'gastritis'. This cleared up in about 2 weeks, and he remained well until November, 1959, some 6 weeks prior to admission, when he became troubled with eructation.

About 4 weeks before admission he began vomiting increasing amounts of liquid vomit, usually first thing in the morning. The vomitus never contained bile, and he was able to eat solid food afterwards. There was no pain, anorexia, or constipation.

On a visit to London he met an old friend, a doctor, who was shocked at his wasted appearance. It transpired that he had lost about 20 lb. in 6 weeks. This doctor sent him to hospital.

Past History.—Congenital idiopathic scoliosis, which had been investigated at the Royal National Orthopædic Hospital.

ON EXAMINATION.—The patient was a thin, cheerful man, who appeared to have lost weight recently, but seemed otherwise well.

General examination revealed no evidence of dehydration. There was a marked scoliosis and a fungal infection of his finger-nails.

Examination of the abdomen revealed well-marked epigastric succussion, but no tumour or viscus was palpable. Bowel-sounds were normal and there was no visible peristalsis.

No other abnormality was found.

INVESTIGATIONS.—

Blood-count and chemistry were normal.

Gastric intubation produced 1 litre of resting juice, containing no bile on the first, nor on subsequent occasions.

FIG. 268.—X-ray photograph from the barium meal, showing the slit-like filling defect at the end of the first part of the duodenum.

Barium-meal examination by Dr. R. Kemp Harper (Dec. 29): "There was no evidence of a gastric lesion but the duodenal cap was persistently distended and barium entered the second part of the duodenum very slowly. There was still a large amount of barium in the stomach and duodenal cap at the end of 2 hours.

"It was difficult to obtain satisfactory views of the point of obstruction, but one film shows this to be a very short, apparently symmetrical, narrow area, which suggests that obstruction is due to a band or web towards the apex of the duodenal cap." (*Fig.* 268.)

It was felt that 'congenital duodenal web' was a reasonable clinical diagnosis, and that operation was necessary.

AT OPERATION (Jan. 2, 1960).—By Mr. Ian P. Todd under general anæsthesia.

The abdomen was explored through a right paramedian incision. The stomach was hypertrophied, with a normal patent pylorus. The first part of the duodenum was dilated up to the junction of first and second parts. No external cause for this was visible. Duodenotomy was performed and an occlusive diaphragm was found, with a central stoma which just admitted the tip of a pair of Dunhill forceps. Bile was seen welling up in the bowel distal to this web.

The web was excised along its circumference and the mucosa was repaired with catgut. The duodenotomy was closed transversely.

HISTOLOGICAL REPORT.—"Specimen consists of a ring of tissue, 1·8 cm. diameter, apparently lined by intestinal mucosa, which encloses a small central orifice 0·3 cm. in diameter.

"Section shows the duodenal diaphragm to be covered by normal duodenal mucosa on both sides. Mild non-specific chronic inflammatory changes are seen in the lamina propria." (*Fig.* 269.)

POST-OPERATIVE COURSE.—This was uneventful and the patient was discharged on Jan. 15. At follow-up one month later he was symptom-free and had regained most of his lost weight.

DISCUSSION

Duodenal diaphragm is very rare in adults. A study of the literature has produced 11 other cases. In some of these the condition was discovered incidentally at autopsy. Only 1 correct pre-operative diagnosis has been recorded, and on occasions the condition has not been discovered even at operation, with a result that the obstruction was not relieved.

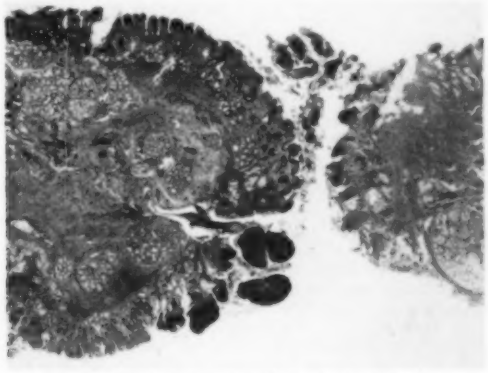

FIG. 269.—Histological section of the duodenal diaphragm, showing the position of the orifice. (× 12.)

Krieg (1937) has reviewed the literature. Some 2 per cent of obstructions in the duodenum are said to be due to a diaphragm. Nevertheless a diaphragm is said to be the only form of intrinsic congenital obstruction which can present in adult life. In an adult, once the obstruction has been localized to the duodenum, extrinsic congenital causes which enter into the differential diagnosis include abnormal fixation and kinking, volvulus of the small intestine, persistent hepato-duodenocolic ligament, annular pancreas, and vascular abnormalities.

An imperforate diaphragm must present early in neonatal life, as do other forms of intrinsic congenital duodenal obstruction. It appears, however, that about half of the recorded cases of diaphragm did not present until after the first year. The 12 cases recorded in adults represent about one-third of those described, though it is probable that this figure is a little high.

The age range of the adult cases described lies between 26 years and 76 years, there being none between the lower limit and 10 years. Sex distribution is even.

The aetiology of this and other congenital intestinal occlusions has been well investigated by Louw (1959). There is said to be an association with other congenital abnormalities.

A pre-operative diagnosis can be made only by radiology. In this case the right diagnosis was suggested to Dr. Kemp Harper by recent articles on prepyloric septa, a somewhat similar condition. In one of these (Rowling, 1959) it was stated that a prepyloric septum may be visualized in any projection.

In this case, however, the septum, being in the duodenum, was seen, as an 'end on' view, in only one film. Duodenal septa, therefore, are not demonstrated as easily as prepyloric septa. Nelson (1947) reported a correct pre-operative diagnosis on radiological grounds.

The treatment of choice seems to be duodenotomy and excision of the web, although in infants a by-pass operation such as gastro-enterostomy is sometimes preferred. Bartlett (1959) reported a successful case in a woman 76 years of age treated by duodenotomy. In his case it was necessary to insert a drain of polyethylene in the ampulla of Vater because of its proximity to the web. Diaphragms have been described everywhere in the duodenum, and the ampulla of Vater has been situated on the web itself.

Presumably an inflammatory episode must be the factor which leads to stenosis of the aperture and the late onset of symptoms, but in the present case histological section shows very little evidence of inflammation.

SUMMARY

A case of subacute intestinal obstruction in an adult due to congenital diaphragm of the duodenum is recorded.

The diagnosis was made by radiography, and the condition was treated by duodenotomy and excision.

Eleven other cases in adults have been recorded. The diagnosis may be missed, even at operation, if the condition is not suspected.

The operative correction is not difficult, but the possibility of the ampulla of Vater opening between the leaves of the web must be borne in mind.

Acknowledgements.—I thank Mr. Ian P. Todd for permission to report this case, and for help and encouragement in its presentation; Dr. R. Kemp Harper for the radiological opinion; Dr. A. G. Stansfeld for the pathological report and the section; Mr. J. A. Cholmeley for a report on the spinal condition; The Department of Medical Photography, St. Bartholomew's Hospital, for the prints; and the Honorary Editors of the *Proceedings of the Royal Society of Medicine* for permission to publish this case reported to the Clinical Section, Mar. 11, 1960.

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REVIEWS AND NOTICES OF BOOKS

Surgical Treatment of Unequal Extremities. By CHARLES WEER GOFF, M.D. 11 × 8½ in. Pp. 184 + x, with 136 illustrations. 1960. Springfield, Ill.: Charles C. Thomas. (Oxford: Blackwell Scientific Publications Ltd.) 88s.

DR. GOFF starts with a review of current knowledge of the growth of long bones with methods of assessment of bone growth, both by means of naturally occurring growth arrest lines and methods used in experimental animals with chemical bone markers. There is an excellent review of radiological methods of accurate length measurement: the method used at the Newington Hospital for Crippled Children involving three exposures of lower limbs, with pelvis level and precautions to ensure parallel beam at each level. The formulæ for estimation of growth in the epiphyses are considered. Dr. Goff favours the method advocated by Green and Anderson, but taking into account, in addition to bone age, body-build, stature, weight, and endocrine status. After discussing the factors leading to growth inequality, treatment of limb inequality is considered. Various methods of growth stimulation in current use and used in the past are considered, and methods of shortening and lengthening legs are discussed fully. Growth control by decreasing epiphysial growth is most favoured. Epiphysiodesis, as advocated by Phemister, is regarded by Dr. Goff as a last resort, in that he prefers stapling to decrease epiphysial growth and describes fully his methods, both when staples are inserted with the intention of removing them when equalization has been obtained (the method he prefers), and staples inserted with the intention of leaving them undisturbed until the end of bone growth. Dr. Goff found that secondary procedures, such as replacement of staples and removal, were more often required than following epiphysiodesis. He had few complications, such as the production of limb deformities, like genus valgum and varum, and those found were easily eliminated. The absolute necessity for careful follow-up after operations upon growing epiphyses is emphasized, and patients are seen after operation at intervals of not more than four months until the end of bone growth or removal of staples. The results described were very good indeed.

Dr. Goff took the opportunity during operations to make a large number of biopsies of epiphysial cartilage, without any ill-effects on the patients concerned. In a total of 72 biopsies from 39 children he was able to correlate the state of affairs before stapling and after 12–96 months of stapling, and compare the histological appearances with those produced in experimental animal studies. Direct compression of staples inhibited the proliferative stage of epiphysial cartilage maturation. The columns of cartilage altered their orientation from longitudinal to horizontal, with steady thinning of the epiphysial disk with time, and eventually tongues of bone crossed the disk and these became stronger until all growing cartilage was eliminated.

In a chapter on fractures in children, Dr. Goff confirmed that the growth stimulation following fractures of long bones in children was proportionate to the severity of the original trauma. Femoral shaft fractures averaged 1·5 cm. of lengthening: this was reached at least by 2 years following injury and was permanent. He had not found a premature fusion of the epiphyses on the injured side. He made clear that there was a good deal of individual variation and could find no method of assessing with certainty the overgrowth in any particular patient. He also confirmed that open operations on limb fractures in children produce the greatest overgrowth.

This monograph is a well-illustrated, careful, objective, and readable review of the whole subject of leg inequality, written by an expert, with a full report of his own results and ideas, and an excellent bibliography.

Handbuch der Urologie, Volume XII. Funktionelle Störungen. By ALFRED AVERBACK, San Francisco; CARL E. BURKLAND, Sacramento; GUSTAV-WILHELM PARADE, Neustadt; J. COSBIE ROSS, Liverpool; DONALD R. SMITH, San Francisco; WALTER W. WILLIAMS, Springfield. 9¾ × 6¼ in. Pp. 312 + xiv, with 74 illustrations. 1960. Berlin: Springer-Verlag. DM.98.

THIS Encyclopædia of Urology is a composite effort by American, British, and Continental contributors. This sort of collaboration is timely, if not overdue, as it will open the eyes of the English-speaking countries to the great strides made, and high standards achieved, by Continental Medicine since the Hitler holocaust.

This volume, devoted to functional disturbances, is perhaps not a prime example of the fruitfulness of integrating the Anglo-American and the Continental medical approach. Thus the latter is solely represented by a dissertation on the urological manifestations of tetany. This may be more comprehensible to the American than to the British reader, since one of the American contributions, on the urogenital effects of allergy, is very similar in conception. The British reader must face up to the possibility that certain subjective urogenital disturbances may be caused by a latent tetany or allergy. This may not accord with traditional British teaching advocating concentration on common and obvious disorders to the virtual exclusion of rare ones, requiring objective evidence before arriving at a diagnosis in organic disease and, above all, eschewing empiricism in therapy.

The remaining contributions are apposite, and will evoke universal approbation: the effect of the psyche on vesical function has always been underrated or too often taken for granted. The subject of impotence is thoroughly ventilated. The urological implications of paraplegia are admirably covered, reflecting and imparting much practical knowledge. The highlight, to the reviewer, is the chapter on male sterility, from America. It is impressively comprehensive, and concludes with a section devoted to artificial insemination. The ethical problem of A.I.D. is at last tackled, and the writer is to be congratulated on the integrity and circumspection which he has brought to bear on this controversial topic.

Production is good, the illustrations being uniformly excellent. There are, however, rather a lot of misprints. For those undertaking to buy all 19 volumes of the Encyclopædia the price of this volume is reduced to DM.78.40, still a very considerable financial outlay.

Cerebral Anoxia and the Electroencephalogram. Edited by J. HENRI GASTANT, M.D., and JOHN STIRLING MEYER, B.S., M.Sc., M.D., C.M. 9¾ × 6¼ in. Pp. 617 + xiii. Illustrated. 1961. Illinois: Charles C. Thomas (Oxford: Blackwell Scientific Publications Ltd.). £9 16s.

THIS is a record of the proceedings of a congress held in October, 1959, in Marseilles, at which the clinical, neurophysiological, neuropathological, and electro-encephalographic aspects of acute cerebral anoxia and hypoxia were discussed. Although the majority of papers were given in French they are here translated into English and are placed in the order in which they were delivered. Some fifty-six contributions are followed by a concluding chapter in which the editors, the one from Marseilles, the other from Detroit, are assisted by Dr. H. Fishgold, of Paris, in summarizing the proceedings. The American editor, who is Professor of Neurology in Wayne State University in Detroit, has also written a Foreword. The list of contributors is far from complete; some well-known authorities are not mentioned, such as W. Grey Walter on electro-encephalography, who writes Chapter 38, and the author of Chapter 46, Etienne Leclercq, of Lille. The bibliography is likewise incomplete and some of the earliest work on the E.E.G. in carotid compression, done in the Burden Institute in 1944 and reported in this JOURNAL (1944, 1947) has been overlooked, particularly the effect of occlusion of the internal jugular along with the common carotid; it is not always realized that digital compression on the side of the neck by compressing both vessels produces a different tracing from occlusion of the common carotid alone. There is no index. It is concluded that whatever the cause of oxygen deficiency to the brain, the characteristic picture of the E.E.G. is one of slow waves of increased amplitude which are first apparent in the frontal region. The book is well reproduced, as we might expect from the distinguished publishing house of C. C. Thomas, and makes a handsome volume containing many important observations on the effects of oxygen lack on the brain.

Year Book of Neurology, Psychiatry, and Neurosurgery 1960/61. Edited by R. P. MACKAY, S. B. WORTIS, and C. SUGAR. 7¾ × 5¼ in. Pp. 584, with 92 illustrations. 1961. Illinois: Year Book Medical Publishers, Inc. (London: Interscience Publishers Ltd.). 68s.

THE three editors of the respective sections of this valuable annual review remain as previously, and one of its chief attractions is their comments on certain of the contributions. The sections are of about equal length, though slightly more space is given to psychiatry than to neurology or neurosurgery. In the diagnosis of intracranial lesions a further tendency is seen for the displacement of ventriculography by angiography which has become safer with experience and the use of improved media. Some recent experimental work has substantiated the view that the formation of the cerebrospinal fluid by the choroid plexuses is a true secretion and not merely a filtration. In the field of infectious disease, orally administered attenuated living vaccine is displacing the earlier use of killed vaccine by injection. The spirochæte recently regarded as a cause of multiple sclerosis has gone the way of the defunct spherula insularis, like it, being in all probability a contaminant. A reduction in the population of the mental hospitals in the State of New York is ascribed to the use of tranquillizers, but an editorial comment suggests that psychiatry to-day is roughly in the same position as general medicine was in 1881. Surgeons will be particularly interested in the report of an analysis of penetrating head injuries in the Korean war in which radical débridement with primary closure of the dura and scalp was the procedure of choice, grafts of either temporal fascia or fascia lata being used to make a watertight closure. The book will be as much in demand as ever among those whose interest is in the nervous system.

Clinical Neurosurgery. Vol. 7. By the Congress of Neurological Surgeons. 9¼ × 6 in. Pp. 278. Illustrated. 1961. London: Baillière, Tindall & Cox. 96s.

THE seventh volume of *Clinical Neurosurgery*, notices of the previous volumes of which have from time to time appeared in this JOURNAL, contains the edited Proceedings of the Ninth Annual Meeting of the Congress of Neurological Surgeons which was held in October, 1959, in Miami, Florida. The book is dedicated to William John German, Professor of Neurosurgery at Yale and chief of the neurosurgical service at the New Haven Hospital. There are fifteen chapters, for the most part dealing with intracranial tumours such as the gliomas, the acoustic neurinomas, metastases, and tumours of the third ventricle,

and with such questions relating to them as disorders of visual perception, epilepsy, scanning with isotopes, the value of radiotherapy, and techniques for the removal of the intracranial meningiomas. In a delightful chapter on the background of neurosurgery, Dr. German gives us some of his experiences along with some practical advice and finally some speculations about the future. Referring to the electro-encephalograph as that "big box of twitching pens", he reminds us that "unlike the agrarian fields, the mind's crop is not notably improved by long periods of inactivity". An added attraction of the book is the discussion in the form of question and answer which is recorded verbatim at the end of each chapter. Like its predecessors, which it matches in size and form, the book is a fine publication in clear, easily readable type and with well-reproduced illustrations. All who are interested in the surgery of the nervous system will wish to possess it.

Tools of Biological Research, 3rd Series. Edited by HEDLEY J. B. ATKINS, D.M., M.Ch. (Oxon.), F.R.C.S. (Eng.), Hon. F.A.C.S. 9 × 5½ in. Pp. 155 + xii. Illustrated. 1961. Oxford: Blackwell Scientific Publications Ltd. 37s. 6d.

THIS is the report of the third and final symposium held on the Methods of Biological Research at Guy's Hospital. The introductory address by Sir Harold Himsworth contains some valuable comments on the instruments of research. The subsequent papers cover a range of subjects, all of which will be useful to clinical research workers in one field or another, and many of the techniques described are already serving clinical investigation. These include ultra-violet spectroscopy and infra-red spectrophotometry, ionization chambers, gas chromatography, and chromosome analysis. Another subject, ion-exchange resins, has a place in treatment. Autoradiography and ultrasonic radiations are also described by experts.

This book is a worthy successor to the two previous volumes, and the editor is to be congratulated on his initiative in organizing three symposia which have been so valuable to those who were privileged to attend, and are now available to all.

Thrombosis and Anticoagulant Therapy. Proceedings of a Symposium held in Queen's College, Dundee, Sept. 29–Oct. 1, 1960. Edited by W. WALKER. 9¼ × 7 in. Pp. 106. Illustrated. 1961. Edinburgh: E. & S. Livingstone Ltd. 17s. 6d.

Symposium on Anticoagulant Therapy. Report of the Proceedings. Edited by Professor Sir G. W. PICKERING, M.A., D.Sc., M.D., F.R.C.P. 8½ × 5½ in. Pp. 284 + xii, with 78 illustrations. 1961. London: Harvey & Blythe Ltd. 21s.

THESE two books cover much the same ground. They are both reports of symposia, the first held in Dundee in the early autumn of 1960, and the second some six weeks later in London.

The Dundee symposium covered current views on intravascular thrombosis. The participants came from Great Britain and eight foreign countries. There is much food for thought for the hæmatologist, the physician, and the surgeon. Of particular interest to the latter are the chapters dealing with anticoagulant therapy in peripheral vascular disease and after surgical and gynæcological operations. For a more specialized group are chapters on the problems of hæmorrhage during and after the use of extracorporeal circulation. The reader will be convinced that an increasing number of patients, particularly those who have suffered myocardial infarction, will be on permanent anticoagulant therapy, and this will bring its own problems for the surgeon. No doctor, whatever his interests in this period of an ageing population, can fail to gain something of value from this excellent symposium. One chapter of the greatest practical value is that by Professor Owren, of Oslo, on the organization of large-scale lifelong anticoagulant therapy; he helped to arrange the symposium and his constructive comments pervade the discussions. The University of St. Andrews deserves our grateful thanks.

The report of the London symposium gives rather more of the history of anticoagulant therapy, and deals more thoroughly with its use in cardiac disease. There are also chapters on anticoagulants in peripheral vascular disease, as prophylaxis against venous thrombosis in injured patients, and coagulation problems in liver disease. The organization of anticoagulant therapy is dealt with by clinicians, pathologists, and a general practitioner, as well as a report on its integration in the Royal Flying Doctor Service in Australia.

The editors and publishers concerned are to be congratulated on making these symposia available to a wider audience so quickly.

Surgery of the Anus, Rectum and Colon. By J. C. GOLIGHER, Ch.M., F.R.C.S. 10 × 7½ in. Pp. 829 + ix. Illustrated. 1961. London: Cassell & Co. Ltd. £8 8s.

THIS is an extensive and exhaustive work on a subject which the author has made his own. Although there are many works on the colon and rectum there is always room for others provided they have information which is of importance to the modern surgeon.

Professor Goligher has written a most interesting volume which is full of practical points and operative techniques. As one would expect, he has concentrated on the treatment of malignant disease of the colon and rectum, and the experience he gained in his early surgical career at St. Mark's Hospital has borne fruit.

The illustrations throughout this volume are excellent and many of them original, which is very refreshing. Several interesting tables showing the resectability rate and resection mortality-rate are included.

Mr. H. H. Nixon has written two admirable chapters on congenital deformities of the anorectal region and megacolon.

An up-to-date series of references is given at the end of each chapter.

This book will take its place as the reference volume on the surgery of the anus, rectum, and colon, both at home and overseas.

The Spinal Cord. By GEORGE AUSTIN. 9¾ × 6½ in. Pp. 532 + xiii, with 363 illustrations. 1961. Illinois: Charles C. Thomas (Oxford: Blackwell Scientific Publications Ltd.). £10 12s.

THIS is a most comprehensive work dealing with every aspect of the surgery of the spinal cord. The author has had the able assistance of ten contributors to this volume, and every feature of the spinal cord is considered. The anatomy and physiology of the spinal cord come under critical review and many original drawings illustrate these sections. A very comprehensive account is given of the arteries and veins of the spinal cord, and the importance of the circulation in various parts of the cord is stressed in association with surgical approach.

The section dealing with tumours is excellent. It is contributed by George Austin himself, and he has called on his large experience of numerous cases and has illustrated his descriptions with superb drawings and radiographs.

The book contains critical references on all important spinal cord problems.

As a reference volume this book could not be bettered, and it is bound to be popular not only with neurologists and neurosurgeons, but also with surgical registrars who wish to know more about the surgical considerations of the spinal cord.

An Outline Guide for the Care of Post-operative Cardiac Patients. By MERLE E. WHITE, R.N. 9¼ × 6 in. Pp. 108, with 14 illustrations. 1961. Illinois: Charles C. Thomas (Oxford: Blackwell Scientific Publications). 48s.

Now that cardiac surgery is practised in many countries the time is ripe for a concise book for nurses dealing with the care of patients who have undergone operations on the heart.

This book is written in a clear, simple style, and all nurses should benefit from reading it. The author describes the equipment used, including emergency equipment for the cardiac case.

A useful addition to the nurses' library.

The Encyclopædia of Sexual Behaviour. By ALBERT ELLIS, Ph.D., and ALBERT ABARBANEL, Ph.D. In two volumes. 7 × 10 in. Pp. 1059 + xix. 1961. London: William Heinemann (Medical Books) Ltd. £7 15s. per set.

THESE two large volumes deal with every facet of sexual behaviour in all the different countries of the world. The whole work is most comprehensive and the very large list of contributors from England, America, Australia, India, Capri, and Bangkok, to mention a few, are all authorities in their own sphere of work. The editors of this work have amply justified the name of Encyclopædia of Sexual Behaviour, and there can be little doubt that the two volumes will become the reference books for many years to come.

The Vesico-vaginal Fistula. By J. CHASSAR MOIR, C.B.E., M.D., M.A., D.M., F.R.C.S. (Edin.), F.R.C.O.G. 8¼ × 5½ in. Pp. 151 + vii, with 53 illustrations. 1961. London: Baillière, Tindall & Cox. 40s.

THERE can be little doubt that patients with vesicovaginal fistulæ are perhaps the most miserable of women. They cannot keep themselves clean, and the odour of urine is apparent to all who come near them.

Such fistulæ are not often seen in this country, but any operative treatment which will bring about a permanent cure is very important.

Professor Chassar Moir, with his matured clinical experience—for he has operated upon no less than 250 cases of urinary fistulæ—is the one person to write a monograph on this distressing condition.

This monograph is beautifully illustrated and the various operative techniques are well described.

Any surgeon who is interested in the cure of this complaint should consult this book. As a reference volume it will take its place as one of the really useful books.

GORDON-TAYLOR MEMORIAL LECTURE

THE Royal College of Surgeons of England and the Middlesex Hospital, with both of which bodies Sir Gordon Gordon-Taylor was so intimately associated, have decided to institute as a permanent memorial to one who did so much to promulgate the ideals of British surgery throughout the world a "Gordon-Taylor Memorial Lecture". It is suggested that this lecture should be given annually, alternately in Great Britain and Australasia. The lecturer will be appointed on the nomination of the President of the Royal College of Surgeons of England, the President of the Royal College of Surgeons of Edinburgh, the President of the Royal Australasian College of Surgeons, and the Senior Surgeon of the Middlesex Hospital. An honorarium will be given to the lecturer—the amount of this being dependent on the sum raised by the Appeal. It is hoped that this will be not less than £2000, and that readers of the BRITISH JOURNAL OF SURGERY will feel inclined to give this Appeal their most generous support.

Subscriptions should be sent to the Secretary, Royal College of Surgeons of England, Lincoln's Inn Fields, London, W.C.2, and it is intended to close the Appeal by June, 1962.

BOOK NOTICES

[The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest to our readers.]

Surgery of Trauma in the Tropics. By R. L. BATTEN. 8½ × 5½ in. Pp. 234 + viii, with 178 illustrations. 1961. London: Edward Arnold (Publishers) Ltd. 35s.

Theory of Shoulder Mechanism: Descriptive and Applied. By A. K. SAHA, B.Sc., M.B., B.S., F.R.C.S. (Eng.), F.R.C.S. (Edin.), M.Ch.Orth. (L'pool), Professor of Surgery. 9 × 6 in. Pp. 107 + xii, with 78 illustrations. 1961. Illinois: Charles C. Thomas (Oxford: Blackwell Scientific Publications Ltd.). 44s.

Psychotherapeutic Techniques in Medicine. By MICHAEL and ENID BALINT. 8½ × 5½ in. Pp. 236 + xvi. 1961. London: Tavistock Publications. 21s.

Klinische Chirurgie für die Praxis. Vol. II, Part 2. Edited by Prof. Dr. Med. O. DIEBOLD, Prof. Dr. Med. H. JUNGHANNS, and Prof. Dr. Med. L. ZUKSCHWERDT. 10¾ × 7¼ in. Pp. 232, with 143 illustrations. 1961. Stuttgart: Georg Thieme. DM.43.50.

Introduction to Anatomy, 1532. By DAVID EDWARDES. English translation by C. D. O'MALLEY and K. F. RUSSELL. 8½ × 5½ in. Pp. 64. 1961. London: Oxford University Press. 15s.

The Shaking Palsy (Parkinson's Disease). A Symposium. By HAROLD ELLIOT, M.D., and BLAINE NASHOLD, M.D. 10 × 6½ in. Pp. 160 + xxii. Illustrated. 1961. McGill University Press. $5.00.

Künstliche Hypothermie für Herzoperationen mit Kreislaufunterbrechung. Part I. **Untersuchungen über physiologische Veränderungen.** Nr. 996. By Dr. MARTIN ZINDLER. 11½ × 8¾ in. Pp. 82, with 17 illustrations. 1961. Koln and Opladen: Westdeutscher Verlag. DM.24.40 kart.

Wiederheretellunschirurgie und Traumatologie. Vol. 6. Edited by M. LANGE. 9¾ × 7 in. Pp. 140 + iv, with 56 illustrations and 7 tables. 1961. Basel and New York: S. Karger. S.Fr.39.50.

Abdominal Operations. By RODNEY MAINGOT, F.R.C.S. Fourth edition. 10½ × 7 in. Pp. 1414, with 1833 illustrations. 1961. London: H. K. Lewis & Co. Ltd. £10 10s.

Congenital Malformations of the Heart. In two volumes. By HELEN B. TAUSSIG. Second edition. Vol. I: **General Considerations.** 10 × 6¾ in. Pp. 204 + xxvi. Illustrated. Vol. II: **Specific Malformations.** 10 × 6¾ in. Pp. 1049 + ix. Illustrated. 1961. London: Oxford University Press. 120s. net (2 vols.).

Essays on the First Hundred Years of Anæsthesia. Vol. II. By W. STANLEY SYKES, M.B.E., M.B., B.Chir. (Cantab.), D.A. 8½ × 6½ in. Pp. 187 + viii. Illustrated. 1961. Edinburgh: E. & S. Livingstone Ltd. 30s.

Cerebral Infarction: The Role of Stenosis of the Extracranial Cerebral Arteries. Medical Research Council, Special Report Series No. 300. By PETER O. YATES and EDWARD C. HUTCHINSON. 9½ × 6 in. Pp. 95 + viii. Illustrated. 1961: London: H.M.S.O. 14s.

Night Calls: A Study in General Practice. By MAX B. CLYNE. 8½ × 5½ in. Pp. 216 + xvi. 1961. London: Tavistock Publications. 21s.

Fellowship Examination Papers for the Diplomas of the Royal College of Surgeons of Edinburgh, 1956–1961. 7 × 5 in. Pp. 55. 1961. London: E. & S. Livingstone Ltd. 5s. 6d.

A History of Thoracic Surgery. By RICHARD H. MEADE, B.S., M.D. 9 × 5½ in. Pp. 933. Illustrated. 1961. Illinois: Charles C. Thomas (Oxford: Blackwell Scientific Publications Ltd.). £11.

The Use of Drawing in the Study of Anatomy and Physiology. By CHARLES R. BANNISTER, M.C.S.P. 11 × 8½ in. Pp. 40, with 19 illustrations. 1961. Edinburgh: E. & S. Livingstone Ltd. 17s. 6d.

Proceedings of the Fourth International Congress on Clinical Chemistry. Edited by C. P. STEWART, P. C. JOCELYN, and S. C. FRAZER. 9¾ × 6 in. Pp. 212 + xvi. Illustrated. 1961. London: Butterworth. 35s.

Alfred Hospital Clinical Reports, Vol. 10. 9¼ × 6 in. Pp. 104. Illustrated. 1960. Melbourne: Alfred Hospital.

The Scientific Basis of Medicine, Annual Reviews, 1961. 8¾ × 5½ in. Pp. 342. Illustrated. 1961. London: The Athlone Press. 40s.

La Grosse Tubérosité de l'Estomac. By T. SCHOPS. 9½ × 6 in. Pp. 574, with 475 illustrations. 1961. Paris: G. Doin et Cie. 125 N.F.

Anatomia Funcional y Cuisurgica de La Mano. By Dr. EMANUEL B. KAPLAR. 10½ × 7½ in. Pp. 326, with 132 illustrations. 1961. Buenos Aires Editorial Artecnica. No price given.

Report No. 2. VIIe Congrès International d'Oto-Rhino-Laryngologie. Vol. 9: Cancer of the Larynx. By J. LEROUX-ROBERT. 9½ × 6¾ in. Pp. 323 + xii, with 18 illustrations. 1961. Basel: S. Karger. S.Fr.36.

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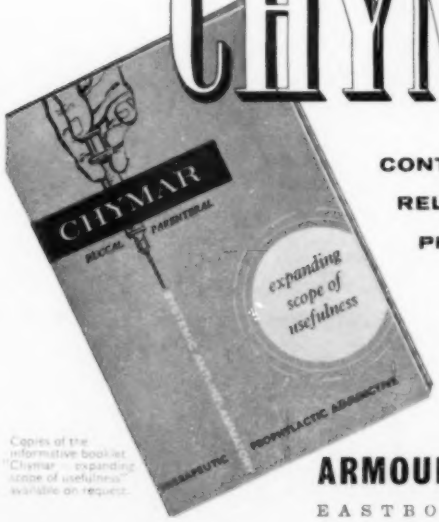

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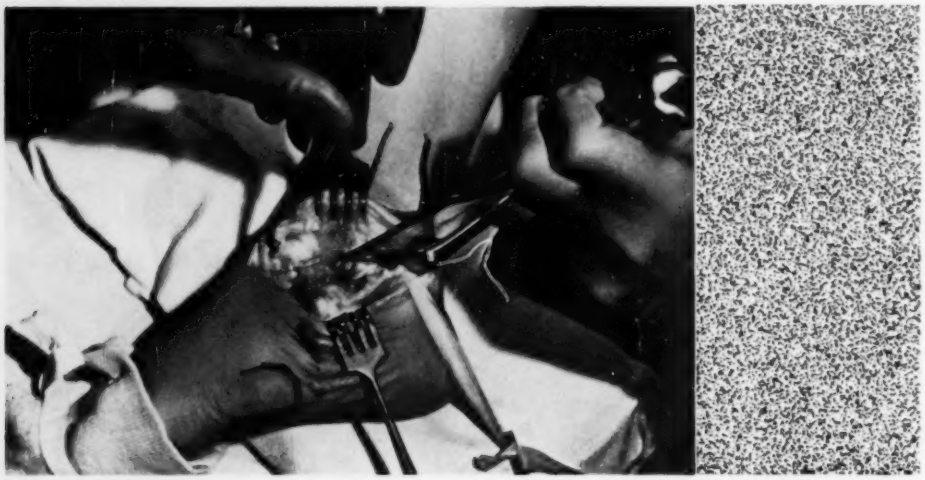

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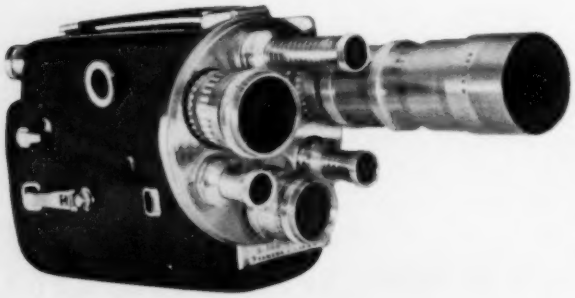

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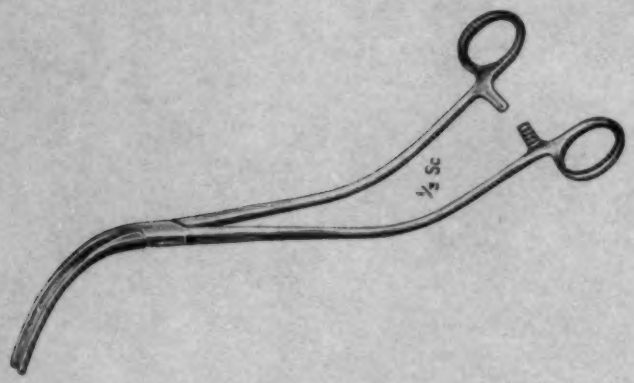

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